



TREATMENT  
OF THE PATIENT  
PAST FIFTY



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TREATMENT  
OF THE PATIENT  
PAST FIFTY

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(OTHER TITLES IN PREPARATION)





FIG 1 —Calcareous aortic stenosis

# TREATMENT OF THE PATIENT PAST FIFTY

*by*

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A RARE EXAMPLE OF YOUTH

IN OLD AGE



## PREFACE TO THE THIRD EDITION

INTEREST IN geriatrics is steadily growing. An increasing number of books concern themselves with the problems of the aged, and during 1946 two new periodicals devoted to the aging process and to diseases of the aged made their appearance. And this volume is now being issued in its third edition. This has provided opportunity for another revision of the text. New material has been added: discussion of temporal arteritis, bacterial endocarditis, acute pancreatitis, fractures of the femur, infections of the respiratory tract, hemolytic jaundice, herpes zoster. The section on mental disorders has been rewritten and expanded. Several new illustrations have been included.

For the color photographs I am indebted to Dr. P. Klemperer, and for some of the roentgenograms to Dr. M. Sussman, both of Mount Sinai Hospital, New York City. I cannot speak too warmly of the unfailing enthusiasm and co-operation of the publishers.

This volume does not pretend to the stature of an exhaustive handbook on geriatrics. It is planned as a simple but complete text to aid the practitioner in the handling of his elderly patients.

ERNST P. BOAS

New York City  
March 1947

## PREFACE TO THE FIRST EDITION

THE MIDDLE AGED, the elderly, and the aged make up a steadily increasing proportion of the physician's practice. The medical practitioner no longer spends most of his time caring for babies with summer diarrhea, for children with diphtheria, scarlet fever and measles, for adults with typhoid fever, malaria and tuberculosis. His young patients are far healthier than they were in former generations, and many more of them grow to middle age and become afflicted in their mature years with circulatory disorders, diabetes, chronic "rheumatism," cancer or some of the other so-called degenerative chronic diseases. The care of these older patients requires an understanding of the psychological and physical involutional changes that take place in the aging person, and of the manner in which these changes influence the manifestations of disease. The diseases to which older persons are subject are insidious in their onset, chronic in their course and lead to irreversible changes in the human organism. The physician must equip himself to understand the aging process and to diagnose and to treat these chronic diseases that are much more complex than the acute infectious diseases that have heretofore occupied so much of his time.

This text has been prepared to serve as a guide to the physician in the management of the aging and aged patient. It

is not intended to give an account of every disease that may affect an older person. The diseases described are those that are common in advanced life or whose occurrence is practically confined to the period of senescence. Strictly speaking, there are no diseases of the aged. Arteriosclerosis and cancer occur in youth as well as in advanced life. But the reaction of the body to disease and the course and frequency of disease differ at different ages. Knowledge of disease is not enough. The physician must distinguish between the process of aging and of disease as it appears in the aging organism. In each case he should determine how far the aging process has advanced and in what respects it modifies the phenomena of disease, as well as the nature and progress of the disease itself.

Senescence does not begin suddenly at a certain chronological age. Most of the chronic diseases of later life have their inception at or before the age of 50. Most deaths occur after this age, and in these older age groups preventive medicine is just beginning to lower the mortality, as it has done so brilliantly in the younger decades of life. Aging actually begins at birth, or indeed at the moment of fertilization of

past 50. In addition to descriptions of disease, the reader will find a discussion of the aging process, as well as consideration of the aging person as a whole, and a presentation of principles for general management of the aging person.

The practitioner of medicine, by studying the natural history of disease in his aging patients and by utilizing in their treatment the fruits of medical investigation and knowledge, is able to defer many of the chronic diseases that are erroneously attributed to old age. Thus he can actively share in the

chief task of medicine during the coming generation—the extension of the human life-span and the preservation of the aging person as a healthy useful member of society

ERNST P BOAS

New York City  
October 20 1911

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*Chapter O***AGING AND DISEASE**

AT THE beginning of the twentieth century 15 per cent of our population were over 65, and 15 45 years of age or older, today 6 8 per cent are 65 years of age or older, and 27 per cent are 45 years of age or older. In 1946 there now are actually 6,000,000 more persons 65 years of age or older than there were in 1946. It is estimated that by 1980 the total number of the 65+ group will be 22,000,000. Two important population trends are the marked fall in the birth rate, which has dropped more than a third in the past 40 years, and a sharp drop in immigration of young adults.

At the same time there has been a great increase in mean life span, that is, in the average expectancy at birth. At the time of the American Revolution the length of life was 35 5 years, by 1900 it had risen to 47 years, and today it is over 65 years. This extension of life span is due largely to the reduction in the death rates in infancy and childhood and in early adulthood. The reduction in death rates has taken place chiefly among individuals below the age of 50, but there has been a saving of life in the older age groups. With the reduction in the ravages of the acute infectious diseases there has been a steady increase in frequency of the chronic diseases.

which today are responsible for three quarters of all deaths. This increasing prevalence of certain chronic diseases, such as arteriosclerosis and hypertension, has been ascribed to the strain and stress of our complex modern civilization. Unprejudiced study, however, demonstrates that the increasing prevalence of chronic diseases is due to beneficent rather than to malign forces. We are spared attack by the infectious diseases in our younger years and survive to succumb at a more advanced age to one of the chronic diseases. Over 60 per cent of all deaths now occur after the age of 45. The physician is called on to treat more and more older patients suffering from one of the chronic diseases.

Cardiovascular renal diseases are the most frequent of all the chronic diseases. In the decade 50 to 59 they are responsible for 40 per cent of all deaths. Their frequency increases with each succeeding year of life, and in the decade 80 to 89 they cause two thirds of all deaths. Among the other chief causes of death in persons past 50 years of age are cancer 12 per cent, accidents 8 per cent, influenza and pneumonia 7 per cent, tuberculosis and diabetes. There are other diseases, common among older persons, that cause much illness and disability but rarely appear as direct causes of death. Most important among these are chronic "rheumatism" or rheumatoid arthritis, the various organic diseases of the nervous system, gastro-intestinal disorders and pulmonary emphysema.

The frequency of various disabling illnesses in the aged can be gleaned from an analysis that was made of more than 500,000 patients discharged from hospitals in New York City in 1933. Table 1, taken from this work, lists the frequency of the chief diseases encountered among hospital patients and their rate of incidence in different age groups of the population. Among patients 65 years of age and over, cardiovascular

diseases have by far the highest incidence. Then follow malignant neoplasms, cerebral arteriosclerosis and hemorrhage, fractures and cataract. Diabetes, bronchopneumonia and arthritis are common and have a rate of incidence higher than in the younger age groups.

Most of the diseases of the aging organism that we have enumerated are popularly considered and termed "degenera-

TABLE 1—FREQUENCY OF DISEASE BASED ON 487,896 HOSPITAL DISCHARGES IN NEW YORK CITY (1933)\*

(Rate per 100 000 population of specified age group)

DISEASE	AGE		
	65 and over	45-64	35-44
Heart diseases	2 535	1 031	337
Vascular diseases	2 110	526	96
Malignant neoplasms	1 049	531	162
Cerebral arteriosclerosis hemorrhage	798	228	30
Fractures	733	404	263
Cataract	724	169	17
Diabetes	563	317	72
Bronchopneumonia	407	144	70
Nonmalignant neoplasms	269	367	487
Arthritis	275	178	97
Tuberculosis	211	298	266
Lobar pneumonia	187	141	114
Acute appendicitis	72	109	153

\*Welfare Council of New York City Hospital Discharge Study (New York City 1942)

tive" diseases. This implies that they are due to the gradual wearing out of the several tissues and organs of the body, that the disease is, in essence, evidence of aging, that it is an inevitable process of senescence. Were this so, we would have to accept the invalidism of aging as part of our inescapable heritage and consider medicine as powerless to halt its progress or to restore the worn out, dying organism. Although the past century has witnessed a tremendous extension of the average span of life, death is inevitable, and we cannot expect an indefinite prolongation of the life span of the human



race The average expectation of life at birth for white females in the United States is 68.9 years Application of present knowledge of medicine and hygiene should make it possible to extend the average length of life to about 75 years Future medical discoveries may extend this by many years

The distinction between aging and disease is not a purely academic one If, for instance, we regard arteriosclerosis solely as a phenomenon of aging, a process that inevitably makes its appearance in the aging arteries of every person, we may well throw up our hands in the conviction that treatment is futile, research fruitless If we regard arteriosclerosis as a disease process, that manifests itself with particular frequency in older persons, we will strive to discover its causes and marshal in its therapy all the knowledge and methods that we have at our disposal As we shall try to show in the chapter on cardiovascular diseases, there is much evidence to prove that arteriosclerosis, as encountered as a cause of disease among our patients, is not a simple senescent process but a pathological one that may eventually be brought under control So it is with many of the other disabilities of the aged

It is important to reach a common understanding of the nature of the aging process Most people think of aging as a terminal event, as a running down or wearing out of the organism, as a final tissue and organ deterioration preceding death This is an incorrect concept Aging is but a part of the whole cycle of life Growth, differentiation, involution are but different phases of the life curve, manifesting themselves at different rates in different structures of the body The milk teeth become loosened and are cast off in childhood, the thymus gland atrophies in early life the ductus arteriosus closes shortly after birth Yet these phenomena result from processes no different from those that cause similar organ changes in the aged As a matter of fact, life

processes and organ alterations proceed at a far more rapid pace in the young than in the old. Minot has pointed out that during old age the tissue and organ changes reflecting the bodily changes accompanying the time curve of life are at their slowest. All of the phenomena of aging do not appear simultaneously. In the same individual, graying of the hair, far sightedness, loss of elasticity of the skin or of the arteries and decalcification of the bones occur at different ages, and among different individuals there is the greatest variation in the time of occurrence of these so called stigmata of aging. When we speak of senescence, and aging of the organism, we arbitrarily segregate the anatomical and physiological phenomena observed in older individuals, and assign to them attributes of degeneration of the organism due to aging.

Aging and death may result from wear and tear of the organism due to external insults, from the accumulation of inhibiting substances within the organism or from a diminution or gradual extinction of the original vital force. Warthin has claimed that old age may be due to gradual weakening of the energy charge set in action at the moment of fertilization of the egg. It has been said that the tempo of aging is dependent less on the degree of wear and tear than on the inborn strength of resistance to this wear and tear. Senescence is but one phase of the continuous development, growth and differentiation of the living organism between the events of fertilization of the egg and death.

It has been shown experimentally that each organ and tissue has its own time curve of aging, and that this time curve depends on three factors: heredity, the external environment, and the internal environment of the body. The most important of these three elements is heredity. Strains of mice can be inbred so that aging occurs uniformly in all

individuals of that strain Furthermore, it has been shown that, comparing different strains of mice, the anatomical changes occurring in the several organs are strain characteristics in their time of occurrence, that is, that various organs age at different rates in different strains of mice We see the same phenomenon in man Longevity is primarily a family characteristic As Oliver Wendell Holmes said

If you are setting out to achieve three score years and twenty, the first thing to be done is, some years before birth, to advertise for a couple of parents both belonging to long-lived families We observe as family traits early graying of the hair or early hypertension or coronary artery sclerosis Aging is primarily a constitutional phenomenon hereditarily determined It has often been suggested that hormones, particularly the sex hormones, may control the aging process Many have claimed that the menopause marks the first sign of aging in women and that there is a similar climacteric in men Both clinical and experimental studies give no support to this view Experimental studies have shown that the several internal secretory glands may accelerate or retard the time curves of involution of various organs but that primarily the effects depend on inherited characteristics There is no master hormone controlling senescence

The variability of the time of occurrence of the individual phenomena of growth and senescence increases greatly with the progress of the years Thus the variability of the age at which the fontanelle closes is relatively small, measured in months, the variability of the age at which the first teeth erupt is greater, at which the permanent teeth appear still greater, and at which menstruation begins still more marked The age at which presbyopia first becomes manifest shows a much greater spread So it may be expected that physical disablement due to aging will appear at widely scattered

ages, say at 50 in some, at age 80 in others. This adds to the difficulty in determining what phenomena are due to disease and what to aging.

All structural changes found in the aged are not signs of senescence. The older the person, the greater his years of exposure to external insults, and the greater the possibility that his body will show scars of these encounters. A generation ago, when tuberculosis was far more widespread than it is today, almost every adult, at autopsy, gave evidence by the scarring of his lungs that he had undergone a tuberculous infection. These scars were not manifestations of aging, but aged individuals, because of years of exposure to tuberculous infection, almost universally had been infected at some time.

Often it is difficult to distinguish between phenomena of pure senescence and those of superimposed disease—a disease process whose development may be favored by the aging of the tissues. Fractures of the hip, through the neck of the femur, are common in old persons. The bone has become rarefied and brittle, the aged person has lost some of his resiliency, balance and coordination, so that he falls more easily, and the weakened bone breaks more readily. Although aging plays its part, the actual fracture is an accident, it does not connote aging. Similar considerations apply to the so called

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sion of  
secre  
tion in the lungs and prevent the expulsion of this material when the lung becomes infected. The element of infection, however, is an accident, not a manifestation of aging.

The human changes most characteristic of aging, bodily changes that are accepted, in the popular mind, too, as evidence of senescence are loss in height, loss in weight, loss of sexual activity, presbyopia, deafness for high tones, graying

of the hair, loss of elasticity of the skin. None of these alterations of the structure and texture of the body are regarded as disease processes, none of them challenge the continuance of life.

It has repeatedly been pointed out that natural death, death from natural decay or from true senility, occurs rarely in man. Autopsies on old people always reveal a pathological cause of death, though no symptoms were observable during life. The most common causes of death in persons over 65 years of age are arteriosclerosis of the coronary, cerebral or peripheral arteries, heart failure, pneumonia, cancer, intestinal tract accidents.

Some practical considerations can be derived from a contemplation of these facts. In dealing with older patients we should always be on the alert to distinguish between disease and aging. We should not carelessly ascribe their symptoms and disabilities to the running down of their bodies. At the same time we should become fully aware of the physical and mental changes that take place in the aging organism and learn how involutional alterations in the structure and function of the body may affect and alter the manifestations of disease. The physician diagnoses a disease, but a sick person.

Disability and illness, no matter at what age they occur, whether in infancy or in the ninth decade, should be regarded as results of disease, a challenge to the diagnostic and therapeutic art of medicine. Only with such an attitude can knowledge advance, only with such an approach can we hope to control and prevent many of the chronic diseases of advancing years that are, as yet, incompletely understood.

This point of view is of particular importance, because of

the changing complexion of disease that has come with the aging population. With the chronic diseases the chief health hazard, the old methods of disease prevention through the agency of public health officials are becoming less useful. The control of diabetes, heart diseases or chronic rheumatism depends on an alert medical profession trained to early diagnosis and appropriate methods of treatment, a public educated to be on the lookout for early symptoms, and the establishment of adequate facilities for diagnosis and treatment. The attempt must be made to keep well each aging individual. The practicing physician will become the chief agent of preventive medicine in the field of chronic diseases.

Public health officials have been slow to learn this lesson. The origin of the modern preventive medicine program may be found in the reaction of social reformers to the intolerable living and sanitary conditions that accompanied the Industrial Revolution. They were concerned with improving the living and working conditions of the laboring class. They emphasized factory inspection, child labor, working hours, wages, housing, water supplies, sewage and garbage disposal. With the coming of the bacteriological era, physicians and bacteriologists replaced the social reformers as leaders of the public health movement and as public health officials. Dazzled by the vision of eradicating disease by controlling its causative agents, public health work took a totally new direction. It concerned itself with water supplies, with sewage disposal and sanitation, with mosquito control, with destruction of rats, with delousing, with antitoxins and vaccines. The milieu in which the sick person lived became of secondary importance. All of these methods of control lent themselves to mass methods of attack—improvement of water supply, pasteurization of milk, even vaccination.

In the past decades recognition that certain infectious dis-

eases do not lend themselves to such mass attack has led, in the case of certain diseases, to a more direct concern with the individual sick person. Witness of this is the tuberculosis program, with the search for early cases and contact infection and the provision of sanatorium and clinic treatment by health departments, the syphilis program, with the public treatment of syphilitics and the search for sources of infection by health department clinics, and finally the free dispensing of serum and chemotherapeutic drugs for the treatment of pneumonia. To implement their incursions into the field of the individual sick, health officers have had recourse to the practice of health education of physicians and the laity. With the infectious diseases coming more and more under control and with the chronic diseases becoming the outstanding health hazard, the attention of public health officials is being directed along new channels. Here the old methods of public health control are no longer useful. The health officer approaches the cancer problem and finds he must first educate the doctor in the early diagnosis and in methods of treatment, he must warn the public to be on the lookout for early symptoms, he must establish cancer clinics as well as cancer hospitals equipped for radiotherapy and surgery. When the health officer attempts to control other chronic diseases, he must develop similar techniques. Although most public health officers still reluctantly enter the field of adult hygiene and chronic disease, and most practicing physicians resent their incursions as an unwarranted intrusion into the private practice of medicine, the bars between preventive medicine and the practice of medicine are being let down.

While medicine is thus attacking citadels of disease that have heretofore been deemed impregnable and treating conditions that have in the past been regarded as the heralds of inevitable death, there are some among us who are advocating,

in certain cases, a policy that is the very negation of the orientation of medicine. I refer to euthanasia—the intentional killing of persons suffering acutely from some supposedly incurable malady. Such a procedure is a complete negation of all that the physician stands for, and can lead only to a regression from the new paths on which medicine is setting out.

The concept of the sacredness of human life has gradually developed through the ages and has become a distinguishing characteristic of modern civilization. At first restricted to the privileged classes, it slowly was extended to include the larger groups of the population, until today it has universal application. To most, it has become an article of faith. Whether or not we accept the sacredness of human life as an ethical principle, we recognize a continuous trend in our thinking, a real evolution of ideas, that has given new significance to this concept in every successive century. In spite of wars, famines, mass murders by autocratic governments, the belief that every individual life is sacred, that it must be preserved has become a basis for much of our modern civilization and government.

If we accept this view, as we must historically, there can be no discussion of the value of preserving or destroying individual life. Life, as such, is an end in itself, and cannot be voluntarily sacrificed, not to ease suffering but to achieve a more universal ideal. Self preservation is the primary concern of the individual; it has become the primary concern of society.

Society entrusts to physicians the preservation of life. The profession of medicine is consecrated to this end. The physician cannot question the relative values of the various methods of treating; else his hand will falter, his art will suffer. If therapeutic success follows a desperate struggle, the physician may be lost by a moment of doubt, by a transitory moment of



of effort. The true physician and successful healer must accept the sacredness of every individual life as a primary article of faith.

Doubts will inevitably arise during the practice of his calling, particularly among the aged. A man has become mentally disorganized, an affliction to himself and to his family. In spite of years of effort he cannot make the needed adjustments yet he has sufficient insight into the hopelessness of his life to ask: "Would it not be better if I did away with myself?" Another is tormented with severe physical suffering to which there seems to be no end. A man awaiting execution in the electric chair develops appendicitis and needs an emergency operation to make him whole again that he may be killed. Another is a brute or a criminal. Then there are the hordes of congenital imbeciles, of the hopelessly insane, who have become institutional wards of society. Some modern states in which the state is the all in all, the individual nothing but a servant of the state, have already proceeded to liquidate many such useless members of society. The physician must brush aside all doubts, all rationalization, and rededicate himself to his underlying faith, to his calling as a preserver of human life.

## *Chapter Two*

### **THE AGING PROCESS**

WE HAVE defended the view that disease in the aged is not part of the aging process, that it represents a pathological condition—a challenge to diagnosis and treatment. There is, of course, an aging process which should be recognized. The clinical manifestations of disease are the results of the reaction of the body to external and internal traumas, and the reaction of the aging organism differs from that of the youthful one. We should endeavor to obtain a clear picture of the physical and psychological changes that characterize this aging organism, because we must deal with it as the setting within which disease runs its course. We must learn the range of what may be called normality for each age period and must search for means by which the slowly aging body may be made to resist the inroads of disease and premature decay.

With increasing age of the organism there is a progressive dehydration of the tissues with a reduction of intracellular fluid, the colloidal systems undergo alterations, elastic tissue loses its elasticity. Some of the outward manifestations of the underlying chemical changes in the body contribute to the characteristic stigmata of aging. Chief among these are loss of weight and of stature. The back becomes bowed with a gentle kyphosis. The skin atrophies and becomes thinned, the subcutaneous fat disappears, there is loss of elastic tissue,

and the skin becomes dry and wrinkled. There is atrophy of the hair and sweat follicles. The hair turns gray and falls out. The teeth become loosened and are gradually lost.

Changes in the eyes are characteristic. Diminution of orbital fat leads to enophthalmos, loss of tone in the muscles and skin causes drooping of the eyelids, both features combined contributing greatly to the facies of aged persons. Arcus senilis, although not confined to the aged, is common in elderly individuals. Finally, presbyopia, loss of accommodation, is one of the most definite evidences of aging. Careful statistical study has shown a genuine correlation between the age of onset of presbyopia and length of life.

Impairment of hearing, particularly for high tones, begins at about age 50 and slowly progresses. It is due to simple atrophy of the nerve and the end organ in the cochlea, and is an almost universal accompaniment of aging.

Parallel with these signs that are discernible by the layman, and that together constitute the picture of the aged person, there are analogous changes in the internal organs. These will be discussed in the chapters devoted to the several organ systems. In their totality, these changes of the organism lead to changes in function. When he is at rest, or under no great physical strain, the bodily functions of the aged person are adequate for his needs. The fasting blood sugar level tends to be slightly higher in the old than in the young. This may be due in part to an increased renal threshold of sugar excretion. Sugar tolerance tests in persons over 65 commonly reveal an excessive rise of the blood sugar level and delay in its recession to the previous level.<sup>1</sup> Both the urea clearance and the concentrating power of the kidneys decline with age, and there is a slight elevation of the blood urea

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<sup>1</sup> Marshall F. W. "The Sugar Content of the Blood in Elderly People," *Quart. J. Med.*, 24: 257, 1931.

nitrogen<sup>2</sup> There is said to be an increase in the blood cholesterol<sup>3</sup> A progressive decrease in the total and vital capacities of the lungs and an increase in the residual air become manifest particularly after the age of 60 These result from alterations in the thorax and spine that limit the mobility of the thoracic cage, as well as from loss of elasticity of the lungs<sup>4</sup> Calcification of the costal cartilages begins in the second decade of life, becomes accelerated at age 20 and continues more slowly after age 40 By the sixth decade the first costal cartilage is about 80 per cent calcified Full calcification occurs about 10 years later in women than in males, in Negroes the process begins earlier in life and reaches its maximum at an earlier age<sup>5</sup> The temperature of the body, the cardiac output, the respiratory exchange compare favorably to similar functions in youth

As soon as the aging body is exposed to unusual or greater strain, we find that the range of response of the various organs is curtailed and that the power of self regulation of the body to maintain a constant internal environment, the function that Cannon calls homeostasis, is lost An aged person does not tolerate excessive cold or heat, he cannot adapt himself to extreme environments His heart functions well when little demand is made upon it, but it cannot respond to the load of greater exertion So it is with all of the bodily functions With age the bodily reserves are encroached on more and more, and gradually these physical limitations circumscribe the range of response and capabilities of the

<sup>2</sup>Lewis W H Jr, and Alving A S "Changes with Age in the Renal Function of Adult Men" *Am J Physiol* 123 500 1938

<sup>3</sup>Rafsky, H A, and Newman B "Cholesterol Studies in the Aged" *J Lab & Clin Med* 27 1563 1942

<sup>4</sup>Kaltreider N L *et al* "The Effect of Age on the Total Pulmonary Capacity and Its Subdivisions," *Am Rev Tuberc* 37 662 1938

<sup>5</sup>Michelson N "The Calcification of the First Costal Cartilage Among Whites and Negroes," *Human Biol* 6 543, 1934

organism Whereas the young person can abuse his body and overtax his strength with few deleterious results, such efforts in the aged soon lead to disaster

The process of repair of tissues after injury is altered in the aging organism It has been shown experimentally that in senescent animals the power of instituting normal repair for epithelium in certain organs is modified or lost Du Nouy has demonstrated that wounds cicatrize much more slowly in older than in younger individuals This has direct bearing on the problem of operations in the elderly

With progressing age there is a gradual change in the natural resistance of the body to infection This may be attributed in part to atrophy of the glands of internal secretion, of the spleen the macrophage system, the bone marrow and the lymphatic tissue structures that are concerned with immune processes Nutritional deficiencies which are so common in the aged particularly inadequacy of certain vitamin stores and of calcium lessen the organism's power to resist infection Pathological changes in various organs, that become increasingly frequent with advancing years, bring about a lessened local or organ resistance to infection Thus pulmonary emphysema favors the development of bronchitis and bronchopneumonia, prostatic enlargement or large cystoceles allow of ready infection of the bladder circulatory impairment in the extremities often leads to serious infection and gangrene Types of infection vary in young and old This may be due in part to an acquired immunity to many diseases and to different chances of exposure to the several infecting agents Rheumatic fever and typhoid fever are rarely encountered among the aged Pulmonary tuberculosis is common, but manifests itself usually as an indolent fibroid phthisis that is often misdiagnosed as chronic bronchitis On the other hand, pyogenic infections, bronchopneumonia and

erysipelas occur with increased frequency among the aged. Erysipelas occurs more commonly in the decade 60 to 70 than in any other, if morbidity is estimated by age specific rates.

The death rate increases 8 per cent each year throughout life following a logarithmic equation. Statistical analysis shows that this effect of age on death rate is due not to the accidental accumulation of infections or injuries but to the loss of resistance to disease as a result of an underlying aging process. If accumulated injuries were the cause of senile mortality, we should encounter occasional individuals living to exceptional ages such as 150 years. The increased death rate, as such, is a manifestation of aging.<sup>6</sup>

With age there is a slow decrement in all of the functions and in the psychological reactions of the body. There is impairment of bodily strength and of swiftness and exactness of motion. But there is great variability in the time of appearance of these changes, and many a superior oldster may in these functions excel the average younger individual. There is a general belief that old persons have lost the faculty of learning new disciplines and new procedures, that they are resistant to new ideas. Psychological studies have thrown doubt on this view, and many believe that the educability of a person does not necessarily decline with age. The liberalism or conservatism of his outlook on life is a determinant as important as his chronological age. Certainly wisdom, a function involving experience and judgment, is preserved in age.

Impairment of memory is universally accepted as a stigma of aging. Memory is a function of attention. The physical weakness of the aged and the assumption by society that they are no longer useful lead to a sense of inadequacy and to a withdrawal of attention. Lack of memory thus is often actu-

<sup>6</sup> Summs, H. S. "The Problem of Aging and of Vascular Diseases." *Science* 95: 183. Feb. 20, 1942.

ally lack of attention Older people are more conservative than the young, they resist changes in their mode of life, they resist new ideas in manners and morals Again this is not necessarily the result of senile changes in their brains and modes of thought, but may result from the sense of inadequacy which their insecure position in society impresses on them, which gives them an emotional need for an unchanged world

Many of the mental and emotional changes manifested in elderly persons are due less to aging and regression of their mental faculties than to the kind of life forced on them by a heedless society Lawton has pointed out that economic and social insecurity plays a large part in hastening mental deterioration in older persons A man who has worked most of his life, who has supported and brought up a family, who has regarded himself, if he ever gave thought to the matter, as a useful member of society is suddenly without work and without income for reasons of age alone In spite of his best efforts, current concepts and customs make it impossible for him to obtain employment and maintain his independence He becomes dependent on his children or on society He learns that he has no further function in life, yet instinctively he clings to life This change in his status, forced on him by the workings of the culture of the society within which he lives, rather than by any cause residing within himself, colors his mental and emotional reactions We draw the unwarranted conclusion that many of the mental and emotional characteristics of the aged are caused by intrinsic biological changes, instead of recognizing that they are the result of external conditions imposed on them by the cultural pattern of the society within which they live The validity of this view is strengthened by the repeated observation of intellectual vigor and emotional balance in individuals who have passed the eighth decade of life and who, because of certain

fortunate circumstances, can continue lives of financial independence and intellectual or creative productivity

An old person who is living a vigorous, useful life regards with resentment the physical disabilities that gradually overtake him. He is annoyed and disturbed that he must now give some heed to his much abused body. The old man who is living an aimless life is much more apt to cultivate his disabilities and to learn to live for them alone. In treating his aging patients the physician should give due regard to these social and psychological problems that so largely determine the patient's reaction to illness and disability. To retard the process of mental senility he should address himself not alone to the task of controlling the progress of organic disease, but of maintaining his patient as a useful member of society. The second part of this undertaking is often far more difficult than the first and will lead the physician beyond the rigid domain of medicine.

A new concept of aging and disease was advanced by the Russian scientist Bogomolets.<sup>7</sup> If confirmed, it may have a far reaching influence on our views of senescence and on the treatment of many chronic diseases. Bogomolets conceived of the connective tissues as a physiological system whose functions are basic to the activities of all organs and to all other processes in the body. It has a *trophic* function, regulating cellular nutrition and metabolism, a *plastic* function that is concerned with the healing of wounds and the regeneration of tissues, a *protective* function related to phagocytosis and the tissue reaction to new growths, an *autoregulative* function resulting in the formation of stimulating substances, especially by the spleen, and a *mechanical* function as an osseous and elastic skeleton. Bogomolets believed that the

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<sup>7</sup> Bogomolets, A. A. "Antitumescular Cytotoxic Serum as a Means of Pathogenetic Therapy," *Am Rev Sov Med*, 1:101, 1943.



physiologic age and the health of the body are determined by the condition of the connective tissues and that the physiologic state of the connective tissues affects all kinds of diseases as well as the process of aging

He developed an antireticular cytotoxic serum which is prepared by injecting into a horse, cells of the spleen and bone marrow of a healthy person who has died suddenly. Large doses of this serum injure the connective tissues, but small stimulating doses are said to stimulate the plastic, protective and trophic functions of the connective tissues and so assist the natural healing power of the body. This serum has had widespread use in Russia, and Bogomolets and his colleagues reported remarkable results in modifying the course of various infections, in preventing recurrences after operative removal of cancers and in accelerating the healing of fractures and of wounds. Bogomolets further suggested that the serum might, by prolonging the physiologic activity of the trophic functions of the connective tissue, furnish a means of postponing senescent changes in the body.

### *Chapter Three*

## **GENERAL MANAGEMENT OF THE AGING AND AGED PERSON**

THERE IS no short cut to the maintenance of health and the prevention of disease in the aging. The diseases to which the aging person is subject are insidious in onset and chronic in course. Their causes are in large measure unknown. Methods for the prevention of disease must be directed along the channels of individual hygiene and periodic examinations designed to discover abnormalities of function and structure at their first appearance and in their earliest stages. To achieve this end the stereotyped periodic health examination performed in a routine manner is insufficient. It is necessary to include a survey of the daily life of the patient, his habits, his mental and psychological make up and his relationship to his wife, his children, his business and his environment. The physical examination should lay special emphasis on the discovery of defects which are known to occur in the aging organism. The physician should always keep in the forefront of his mind that it is his primary function to preserve the aging person as a useful member of society, with a normal relationship to the world about him. In his enthusiasm to treat specific abnormalities, the physician is apt to overlook the fact that a too drastic change in the patient's mode of

life may have unfavorable psychological consequences that far outweigh the benefit that may be derived from the proposed regimen. When specific medical measures are life saving there is no choice. When the issue is one of balancing the value of retirement from work, and dedication of the patient's whole life to the preservation of his health, against some compromise plan by means of which the patient may still feel that his term of usefulness is not yet ended, the physician should always endeavor to work out a plan that will preserve the patient's individuality and self-respect. Far too often, doctors recommend complete retirement, uproot the patient from the community where he has spent all of his life and send him to a place like Florida or California. No generalization can be made in these matters, but in every case the physician should keep in mind that he is treating a person and not a disease. In the treatment of acute diseases, such as pneumonia, or acute surgical conditions this factor is of relative unimportance, but in the adjustment of a patient's life in the endeavor to retard the progress of a chronic illness social and psychological factors loom large. Indeed it is probable that a suddenly enforced retirement; the resulting idleness and sense of uselessness the overwhelming realization that the life one has lived and striven for is finished, may hasten the progress of disease and even favor early death. It is probably more than a coincidence that one reads so frequently of the sudden death of a sea captain for instance, a few months after his ret -

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or the old man can still do light chores on the farm or similar tasks in the home. Thus, the transition can be made without the shock that a complete scrapping or a discarding of the aging individual inevitably brings about.

In treating aging persons, the physician should give constant consideration to the loss of youthful resiliency and adaptability of their tissues and organs and to their progressively lessened range of response to the calls made on them by the demands of daily living. The young person can abuse his body almost with impunity; he can exercise to complete fatigue; he can permit himself excesses in eating and drinking, he can smoke too much, he can go without sleep. His recuperative powers are great, and rarely does he do himself permanent damage. But when middle age is passed, the body can no longer adapt itself so readily to extreme stresses; soon some structure or function gives way and leads to the beginning of disease. Thus, with advancing years, the individual must learn to know his increasing limitations and arrange his living so that he does not overtax the weakening organism. Here again one should try to strike the balance between the maintenance of physical fitness and excesses that lead to overstrain.

### NUTRITION

There exists a balance between the food requirements of the body and its rate of growth and degree of activity. Food requirements are in reality energy requirements, for food provides the source of growth and energy. Thus changing expenditures of energy are paralleled by change in the quantity of food needed. The caloric needs of the growing child per kilogram are far greater than those of the adult, of the laborer greater than of the sedentary clerk. Most of us, as we pass middle life, involuntarily manifest a slowing down and diminution of our physical and sexual activities. But

appetite and food consumption are largely matters of habit, they are not automatically adjusted to the actual caloric needs of the body. So it commonly happens that, after the age of 40, food intake is in excess of the energy requirements of the body, and adiposity results. Increase in adipose tissue and the development of the panniculus adiposus with the resultant pot belly are almost universal in middle age. It is only when the seventh decade is passed that a loss of weight occurs, due in large measure to loss in water content of the tissues.

With increasing age there is a gradual lowering of the basal metabolic rate. At the age of 60 the reduction is about 10 per cent, and in extreme senescence it may reach 25 per cent. This lessened oxygen consumption is caused in part by involution of the thyroid gland, in some measure by diminution in muscular vigor. It contributes to the lessened food requirements of the aging body.

The caloric needs of a man living a quiet life indoors are about 2,200 calories, of one engaged in a sedentary occupation 2,500 calories, and of one leading a more active life 3,000 calories. A worker at heavy tasks, such as carpentry, needs about 4,000 calories, and one engaged in exceedingly laborious work such as lumbering 6,000 calories. It is evident that with advancing years and lessened physical activity the caloric needs diminish. Absolute figures cannot be set down for each age group, but the principle is clear that as a person grows older his food intake should be cut down. Most persons past 70, who live very quiet lives, should consume between 1,500 and 1,800 calories daily.

The protein needs of the average adult are placed at about 75 Gm a day. The aging body needs less protein and is less able to handle an excess of ingested protein. The protein intake should be reduced in proportion to the reduction in caloric intake. Thus an old man whose requirements are

TABLE 2.—WEIGHT—HEIGHT—AGE

Age	5	5.1	5.2	5.3	5.4	5.5	5.6	5.7
40	131	133	135	138	141	145	149	153
41	131	133	135	138	141	145	149	153
42	132	134	136	139	142	146	150	154
43	132	134	136	139	142	146	150	154
44	133	135	137	140	143	147	151	155
45	133	135	137	140	143	147	151	155
46	134	136	138	141	144	148	152	156
47	134	136	138	141	144	148	152	156
48	134	136	138	141	144	148	152	156
49	134	136	138	141	144	148	152	156
50	134	136	138	141	144	148	152	156
51	135	137	139	142	145	149	153	157
52	135	137	139	142	145	149	153	157
53	135	137	139	142	145	149	153	157
54	135	137	139	142	145	149	153	157
55	135	137	139	142	145	149	153	157
56	135	137	139	142	145	149	153	157
57	135	137	139	142	145	149	153	157

## WOMEN

Age	4.8	4.9	4.10	4.11	5	5.1	5.2	5.3	5.4	5.5	5.6	5.7	5.8	5.9	5.10	5.11	6
40	119	121	123	125	127	129	132	135	138	142	146	150	154	158	161	164	167
41	120	122	124	126	128	130	133	136	139	143	147	151	155	159	163	166	169
42	120	122	124	126	128	130	133	136	139	143	147	151	155	159	163	166	169
43	121	123	125	127	129	131	134	137	140	144	148	152	156	160	164	167	170
44	121	123	125	127	129	131	134	137	140	144	148	152	156	160	164	167	170
45	122	124	126	128	130	132	135	138	141	145	149	153	157	161	165	168	171
46	123	125	127	129	131	133	136	139	142	146	150	154	158	162	166	169	173
47	123	125	127	129	131	133	136	139	142	146	150	154	158	162	166	169	173
48	124	126	128	130	132	134	137	140	143	147	151	155	159	163	167	171	174
49	124	126	128	130	132	134	137	140	143	147	151	155	159	163	167	171	174
50	125	127	129	131	133	135	138	141	144	148	152	156	160	164	168	172	176
51	125	127	129	131	133	135	138	141	144	148	152	156	160	164	168	172	176
52	125	127	129	131	133	135	138	141	144	148	152	156	160	164	168	172	176
53	125	127	129	131	133	135	138	141	144	148	152	156	160	164	168	172	176
54	125	127	129	131	133	135	138	141	144	148	152	156	160	164	168	172	176
55	125	127	129	131	133	135	138	141	144	148	152	156	160	164	168	172	176

filled by 1,500 calories a day should reduce his protein intake to about 45 Gm

### WEIGHT REDUCTION

The maintenance of an optimum weight in his patients is one of the primary concerns of the physician, particularly in those past middle age. Life insurance tables have demonstrated that after age 35 overweight is associated with a high death rate and that after 45 overweight is a serious health hazard. Table 2 presents the optimum weight for men and women and can be used as a rough guide. Such figures can not be used arbitrarily for some persons have heavier bones and bodily frames than others, so that their normal weights are greater than those of the average.

In most instances satisfactory weight reduction can be achieved without adherence to a rigid written diet. It is much better to explain to the patient the principles of nutrition that he is to follow and to work out with him the modification of his diet that will achieve the desired end. It is astonishing how relatively small dietary adjustments may bring about a sufficient loss in weight. The first step is to reduce the intake of carbohydrates—bread, potatoes, rice, noodles, cake and pastry and the like. Often this is all that is necessary. In other cases reduction in the size of the portions is effective. The patient is instructed to leave the table a bit hungry, rather than uncomfortably full. Eating between meals—taking soft drinks, ice cream, candies or a snack before retiring—add greatly to the caloric intake. Alcohol has high fuel value and a few cocktails or high balls may add several hundred calories to the daily consumption. Beer, too, is of high caloric value. Bacon and other fatty foods, foods prepared with olive oil or butter, mayonnaise, large helpings of butter can be

easily eliminated. Most patients can lose weight satisfactorily by such an adaptation of their diet, planned with understanding in cooperation with their physician. It is unnecessary to attempt to induce a rapid drastic reduction in weight. It is better for the patient to lose from 1 to 2 lb. a week consistently and in the meantime become habituated to his new

TABLE 3 --1,200 CALORIE DIET

BREAKFAST	
Fruit	1 serving
Bacon	1 slice, crisp
Egg	1
Bread	$\frac{1}{2}$ thin slice
Butter	1 square
Cream	1 tablespoon
Coffee or tea	
LUNCHEON	
Meat	1 large serving
Vegetable	2 small servings
Butter	$1\frac{1}{2}$ square
Fruit	1 serving
Milk (skimmed)	1 glass
DINNER	
Meat	1 large serving
Vegetable	1 small serving
Salad vegetable	1 small serving
Butter	$1\frac{1}{2}$ square
Fruit	1 serving
Milk (skimmed)	1 glass

dietary plan. Such a procedure is much more apt to lead to permanent nutritive balance. To a large extent, habit governs the nature and quantity of food consumed. For the first few weeks the patient may complain of hunger, but he soon becomes accustomed to the new regime and can continue it with perfect comfort.

There are some who do not respond to such simple measures. Some patients cannot be made to realize the importance of an adjustment of their dietary habits. Such persons



must be given the bald facts of the increasing mortality that accompanies overweight, and the whole situation must be dramatized for them. In these cases it is wiser to prescribe a formal written dietary. The patient may be maintained on a 1,200 calorie diet until the desired weight reduction is achieved. Then certain increases may be allowed in order to maintain the ideal weight. There are patients who do not lose weight in spite of rigid adherence to a limited diet. Often this refractoriness is due to water retention. This is favored by excessive ingestion of salt or of sodium bicarbonate. The retained water can be driven off through the kidneys by occasional intravenous or intramuscular injection of a mercurial diuretic, such as mercupurin or salyrgan. The patient diets strictly

the mer . . . low disease, and this weight loss will be maintained. The diet is continued, and every week or two another injection is given until the desired result is achieved. Before giving such mercurial diuretics the physician should make sure that there is no serious kidney disease that may be aggravated by the mercury. A trace of albumin or a rare cast is no contraindication, but in the presence of true renal impairment, signaled by low specific gravity of the urine, the drug is best avoided.

Obesity is rarely caused by endocrine disorders,<sup>1</sup> and the administration of thyroid is usually unnecessary. When the measures already outlined do not succeed it is well to determine the basal metabolic rate of the patient. Readings ranging from minus 10 to minus 20 do not indicate a true thyroid deficiency unless there is an accompanying elevation of the blood cholesterol. However, in such cases the administration of desiccated thyroid may be helpful. It should be given cautiously, with the patient under regular observation. It is

<sup>1</sup>Newburgh L. H., and Conn J. W. "Obesity," *Physiol Rev.*, 24:18, 1944.

well to begin with 1 gr of desiccated thyroid daily and to increase the dosage gradually to a maximum of 3 gr if no weight reduction is obtained. Symptoms of thyroid intoxication are an increase in pulse rate, increasing nervousness, palpitation and sleeplessness. If such symptoms supervene, the drug should be discontinued.

Even in the old, obesity may be conditioned by a psychoneurosis. Many people claim that unless they eat at frequent intervals they feel weak and are overcome by fatigue. They are miserable unless they are constantly eating. Actually this is a mechanism that momentarily assuages the anxiety or relieves the loneliness that evokes the feeling of weakness. Weight reduction may be unsuccessful without treatment of the underlying maladjustment or psychic disturbance.

### DIET

Diet is not all a matter of calories and the maintenance of an ideal weight. Experimental studies in nutrition have demonstrated an intimate relationship between the qualitative composition of the diet and health. They have shown that inadequate diets may cause disease and that the duration of life of individual animals and the health of successive generations can be affected at will by alterations in the diet. One should distinguish between a minimum adequate diet and an optimum diet. For instance, rats kept on a suboptimum diet develop and grow normally, but if this same diet is continued for several rat generations, a gradual deterioration of the succeeding generations takes place. Sherman has shown that prolongation of the period of the prime of life, and an actual extension of the span of life, may be brought about in rats kept on an optimum diet. In his words 'Preservation of the characteristics of youth, and extension of the prime of life

are very significantly aided by the taking of a liberal proportion of the needed calories in the form of protective foods." McCay has demonstrated that retardation of the growth of rats by reducing the calories of an otherwise complete diet will delay and slow the onset of maturity and will greatly prolong the life span. He believes that retardation of growth affords a method of retarding senescence and of extending the life span. Cataracts have been produced in rats by diets high in lactose or galactose (lactose liberates galactose in the body) and low in fatty acids and protein. The common occurrence of cataract in patients with diabetes another condition with perverted sugar metabolism is well known.

Whereas these experimental data cannot be directly carried over to the field of human nutrition, they yield significant leads. The results of inadequate dietaries may be slowly cumulative and may not become apparent for many years. Many of the manifestations of aging and disease in older persons that today are accepted as inevitable results of senescence may well be the result of life long faulty dietaries. Sherman has repeatedly emphasized that the ordinary American diet is more deficient in calcium than in any other element. As a result adults experience a steady, long continued loss of calcium through the years. This does not become superficially manifest because the great calcium stores in the bones constantly make up for the calcium losses in the blood and tissues. But as a result of this constant depletion the bones become poor in calcium and more fragile. Osteoporosis of the aged may be the result of a dietary fault and not an inevitable accompaniment of aging. An intake of almost 1 Gm of calcium a day is needed to maintain the calcium balance of the body. Most foods are poor in calcium. The best sources of this mineral are milk which contains about 1 Gm to the quart, cheese and green leafy vegetables.

Snapper<sup>2</sup> has pointed out that arteriosclerosis occurs rarely in northern China, in spite of the fact that diabetes is common. He suggests that fundamental differences in dietaries may underlie this phenomenon. The Chinese diet contains only small amounts of cholesterol but considerable quantities of unsaturated fatty acids, especially linoleic and linolenic acids. He states that the average cholesterol content of the blood of the Chinese is lower than that of Westerners and suggests that this may account for the lessened incidence of lipid infiltration of the arterial wall among the Chinese.

As yet we have no knowledge of a specific relationship between individual vitamins and aging, but in the light of the experimental work referred to in the previous paragraphs it is evident that the provision of an adequate supply of vitamins throughout life will help to maintain health and probably postpone some of the disabilities that come with advancing years. We have learned to recognize the more extreme manifestations of vitamin deficiencies and have become aware that vitamin deficiency may give rise to disease under unexpected circumstances. Today we know that symptoms of beriberi, due to lack of thiamine hydrochloride, are common in alcoholics who eat little for many weeks and derive most of their calories from alcohol. We see symptoms of pellagra in persons who eat inadequately or who have serious gastro intestinal disease preventing absorption of nicotinic acid and riboflavin. At times we see scurvy in patients with peptic ulcer who are kept too long on a rigid Sippy regimen. Some of the mental disturbances of the aged have been shown to be pellagra like, and have been cured by the administration of nicotinic acid. It is not at all unreasonable to suppose that many of the lesser disturbances that develop with aging are ascribable to die-

<sup>2</sup> Snapper, I. *Chinese Lessons to Western Medicine* (New York City: Interscience Publishers, Inc., 1941), p. 160.

tary deficiencies. Often a primary disease, such as a heart disease, will interfere with the appetite and with the absorption of food so that secondary symptoms due to dietary deficiencies become manifest. In older persons we may encounter dietary fads and idiosyncrasies that lead to disease.

Thus in planning the diets of the middle aged and elderly we should concern ourselves not with calories alone, but with the composition of the diet to insure an adequate representation of foodstuffs containing the protective vitamins and the needed minerals. Vitamin A is found particularly in milk, butter, eggs, cheese, liver and the green leafy vegetables; vitamin B in whole wheat bread, beef, mutton and many fruits and vegetables; vitamin B in milk, fruits and the leafy vegetables; vitamin C in fruits especially oranges, lemons, lemons, tomatoes and melons.

Cheilositis, the macerated lesion at the angles of the mouth, has been considered pathognomonic for riboflavin deficiency. Recently it has been shown<sup>1</sup> that this lesion in older persons is often due to ill fitting artificial dentures with too short vertical dimension, causing the upper lip to overhang the lower. This allows the saliva to escape at the angle of the mouth and creates a moist pocket where fungi and bacteria grow on the epithelial debris. The lesion is more fissured and granulomatous than that caused by ariboflavinosis. It disappears when proper dentures are provided. In true ariboflavinosis there are accompanying lesions of the lip.

Another handicap that must be overcome in planning diets for the aged is the impairment of chewing power due to loss of teeth and to diseased teeth. It is most important to see to it that every aging person has dentures that will enable him to masticate an adequate varied dietary. An old person com

<sup>1</sup>Ellenberg M. and Pollack H. "Pseudoarboflavinosis." J. A. M. A. 119: 90 July 4, 1947.

monly restricts his dietary to cereals, soft bread and tea and coffee because he cannot chew most of the food that is set before him

With age, too, there is apt to be more difficulty in digestion. There is, as a rule, a diminution in gastric juice, particularly in hydrochloric acid, there may be motor disturbances of the stomach and intestines, constipation and the accumulation of gases are common. These, in turn, lead to inadequate absorption of food from the gastro intestinal tract and to dietary idiosyncrasies because of the patient's attempt to correct the gastro intestinal distress.

Thus the diet of each person should be individualized with due reference to the factors that have just been discussed. Its quantity should be adjusted to the correct caloric needs of the individual, its composition should be sufficiently varied to provide not a minimum but a surplus of the necessary vitamins and minerals, its consistency should be adapted to the masticatory power of the patient, and, finally, it should be adjusted to the digestive peculiarities of the individual. As a general rule, a varied diet including fruits, meats, fish, vegetables particularly the green leafy varieties, eggs and milk should serve the purpose. One of the most difficult tasks is to provide an adequate calcium intake, i. e., almost 1 Gm a day. If the patient will drink 1 qt. of milk a day the problem becomes simple. If he will eat liberally of the leafy green vegetables and some cheese daily, the amount of milk can be reduced.

In many cases it is necessary to supplement the diet with some of the vitamins. The most important ones for elderly persons are vitamin A and vitamin B complex. Calcium may be given as calcium lactate 15 gr. three times a day or calcium gluconate 30 gr. three times a day. Forty grains of calcium lactate or 75 gr. of calcium gluconate contain as much cal

cium as 1 pt of milk. Calcium is best given on an empty stomach at least three hours after the previous meal.

Diminution in the sense of taste comes with aging. This may interfere with appetite and may make it difficult to persuade an older person to eat a greater variety of food. He becomes satisfied with a simple, monotonous diet. Condiments may be allowed freely unless some specific disease condition contraindicates their use.

In order to avoid overtaxing the digestive apparatus, it is best to plan for three meals of almost similar size, instead of making the evening meal the heaviest one. Indeed it is wise to have an elderly person partake of his main meal in the middle of the day and take only a light supper. It should be habitual to rest for an hour or so after each meal. This aids digestion and avoids overtaxing the heart. Both the digestive process and muscular activity call for an increased blood flow, and the aging heart may not be competent to meet both needs simultaneously without damage to itself.

Tea and coffee, one or two cups daily, may be freely allowed. These beverages are useful mild circulatory stimulants and diuretics. They rarely do harm unless there is a personal idiosyncrasy to them. In some persons coffee causes digestive disturbances, in others extrasystoles. Coffee may cause wakefulness when taken with the evening meal.

Alcohol in moderation is very useful to the aged. It serves as an appetizer, and in cachexia and malnutrition is helpful because of its food value. Reflexly it acts as a dilator of the peripheral blood vessels and promotes a sense of warmth and well being. Most valuable is its depressant action on the central nervous system. Taken when the subject is worried, tense or overtired, it promotes relaxation and rest and may induce sleep. A glass of sherry or whiskey or some wine taken in the late afternoon after a fatiguing day is very beneficial,

and a drink taken before retiring may promote sleep. Of course, it should be taken in moderation. The *Regimen Sanitas* of Salerno written some 800 years ago, sums up the case for alcohol

Sound wine revives in Age the heart of youth,  
While poor wine acts the other way, forsooth  
Pure wine on all refreshment will bestow  
In brain and stomach cause a cheerful glow  
And stagnant currents force anew to flow  
'Twill all depressing carking gloom remove,  
Sharpen the mind and also sight improve  
Quicken the ear and the whole body nourish  
And cause old age with youthful bloom to flourish

### REST AND EXERCISE

It is difficult to strike a balance between rest and exercise in the aging individual. Rest is certainly better than uncontrolled exertion, but measured and regulated bodily activity has positive physical as well as psychic benefits.

Although the movement of the blood finds its origin chiefly in the cardiac muscular pump, the regulation of the circulation is determined by many additional structures and functions such as the vasomotor tone, the distribution of blood in the arteries, capillaries and veins, the blood volume, the respiration and the action of the voluntary muscles in milking the blood in the veins toward the heart. It is common knowledge that physical training enables an individual to undergo severe bodily exertion with greater ease and efficiency. A series of complex physiological adjustments makes this possible. Through the development of the voluntary muscles and their increased coordination through the agency of the central nervous system, movements are executed with less effort, useless motions are eliminated, and less oxygen is needed by



the body to accomplish an equivalent amount of work. The chest expands, the vital capacity becomes greater so that pulmonary ventilation is more readily increased. The heart muscle, like the striated muscles, undergoes some hypertrophy and has greater contractile power. The nervous regulation of the heart and blood vessels is more coordinated, and there is a closer adaptation of cardiovascular response to the needs of the body. The pulse rate in the trained individual is slower, and since the minute volume flow of blood is unchanged the output per beat is greater. The blood pressure is lower. The oxygen carrying power of the blood may be increased, and there is better oxygen utilization by the tissues. All of these factors give the body a greater range of response to exercise. The increase in pulse rate, minute volume flow of blood and blood pressure induced by exercise is less in the trained than in the untrained.

These circulatory reactions and adaptations to exercise and training apply to elderly persons as well as to young athletes. While there is no thought of making a trained athlete out of a man advanced in years, a moderate amount of well tolerated activity serves to make the circulatory apparatus a more efficient machine, spurs the heart a certain amount of work ordinarily carried by the "peripheral circulation" and promotes a general sense of well being. It also helps to prevent obesity which follows a too sedentary life and is an added burden to the heart.

These considerations lead to the conclusion that in persons past the age of 50 one should aim to maintain physical fitness without permitting too strenuous activity. Sudden spurts of effort, as in lifting heavy objects or sprinting to catch a train, are particularly hazardous, for they may induce acute heart failure. Similarly, all competitive sports in which the sense of rivalry makes the participant forget his fatigue and drives

him to greater efforts should be prohibited. It is difficult to formulate absolute rules. A man who has labored physically all his life can often continue at his occupation into his seventh decade, although one encounters few such men who continue at their work with undiminished vigor after the age of 60. But in such men, too, it is wisest to avoid sudden severe strains, as in lifting. The average man who leads a more or less sedentary life had better give up the more strenuous sports such as tennis and handball, particularly if he plays only occasionally. There are men who continue playing tennis until they are well up in their sixties, but they are exceptional. A good deal depends on the intensity with which the game is played. Golf is well tolerated by most aging individuals, and moderate swimming, too, is permissible. Dancing places a much greater strain on the heart than is generally recognized. It is not unusual for the heart rate of normal young individuals to rise to as high as 120 to 150 beats a minute after three or four minutes of dancing. Walking, hiking, golfing, horseback riding are probably the best activities for the person past 50. The housewife, too, should gradually give up the more strenuous aspects of her work, such as scrubbing floors and doing heavy laundry. The best guide to the degree of activity that may be allowed is the reaction of the individual. Undue fatigue, dyspnea, precordial distress are all signs that the individual is overtaxing himself. Above all, it is important that a man who has lived a completely sedentary life undertake new physical activities gradually and slowly try to get himself into better physical trim. It may be well to repeat that it is the unexpected, sudden, severe physical strain that is apt to damage the aging body.

Stair climbing, traditionally, is a great bugaboo to elderly persons as well as to those with heart disease. Stairs are not

nearly as dangerous or harmful as we make our patients believe. Persons with good cardiac reserve may climb stairs freely, but unhurriedly, it does them no harm. Running up, two steps at a time, is unwise.

We should teach the middle aged and elderly person to observe unhurried, free activity and with it a sense of leisure. It is the everlasting rush from one place to another, from one task to the next, that is wearing and that causes undue strain.

### CLIMATE AND WEATHER

There is no difference in the longevity of people living in different climates, so that climate alone cannot be credited with a determining influence on the aging process. It is true that old people do not stand the cold well, and many of them have emphysema and recurrent attacks of bronchitis that are induced and aggravated by the changeable weather of our north temperate climate. For such, a winter residence in the South, in Florida or in California, is often beneficial. However, only the minority can avail themselves of this resource.

Temperature regulation of the body is less efficient in the aged than in young persons. Elderly persons should avoid extremes of heat and exposure to the sun. The mortality rates from heat stroke are 8 per 100,000 at ages 70 to 79 and 20 per 100,000 at ages 80 to 89. The aged suffer heat stroke or sunstroke more readily than do younger persons. The sensitivity of the aged to cold is well known. Institutions for the aged must maintain a higher temperature in their rooms than institutions harboring younger persons. A temperature of 75° F. is usually gratefully experienced by an old person. During the winter the environmental temperature of the aged should be maintained at a comfortable warmth. For the same reason the aged should dress more warmly than younger people.

When they go out their movements are slow, their physiologic responses sluggish, they cannot generate enough heat through muscular action to compensate for exposure to freezing temperatures. So it is well for old people to wear warm woolen underwear and warm socks and to bundle up in warm garments when they venture out in frigid weather. Warming the bed at night or the use of a hot water bag contributes to their health and comfort.

It is unwise for men in the sixth and seventh decades of life to undertake severe physical exertion in the cold. Cold reflexly may cause narrowing of the coronary arteries, and this combined with heavy work may induce coronary insufficiency and cardiac infarction. Every year one reads of men past the prime of life experiencing heart attacks while shoveling snow or while struggling through a blizzard. I see at least half a dozen such cases annually.

In bathing, too, the aged person should avoid extremes of hot and cold. Baths that are too hot are enervating and weakening and may cause collapse. Cold baths may be too great a shock to the heart and circulation, and in an arteriosclerotic person may cause coronary insufficiency.

### SLEEP

Most persons need less sleep as they grow older. The normal adult usually sleeps some eight hours every night. Many find their need for sleep gradually reduced after the age of 50 and may do well with six hours of sleep. It is claimed that after the age of 65 many individuals sleep only four hours out of the 24, but this must be exceptional. Many persons, as they grow older and feebler, sleep more, as much as 12 hours a night. The duration and character of sleep, even in youth, is a highly individual characteristic, varying

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10 hours Whereas the younger person can voluntarily curtail his hours of sleep for a while, the elderly individual can not do so without showing signs of fatigue and inefficiency. Slowness of recuperative power and lack of elasticity and adaptability of all physiological functions are the major accompaniments of aging. The aging individual cannot abuse his body, as can the youth without paying the penalty of increasing the inefficiency of his involuting body. Many old persons spend a full eight to 10 hours relaxed in bed and are satisfied if they sleep only part of the time. Others continue to go to bed at the hour to which they were accustomed when they were younger and, needing less sleep, awake early and complain of insomnia. Such persons are helped by postponing the hour of retirement.

An afternoon nap has great restorative value and should be adopted as a routine by most aging persons. A short sleep, 15 to 30 minutes is better than a long one. With prolonged sleep there comes such complete muscular relaxation that awakening is difficult, and the individual often feels more tired than before he fell asleep. After the age of 70, especially in persons with progressive enfeeblement, the need increases for short naps throughout the day. Such persons often fall asleep while sitting in a chair, reading or knitting. The sum of all such naps may amount to several hours and compensates in part for the shorter nocturnal sleep.

Insomnia, when it is unrelated to the aging process as such, should be treated as in younger patients, by attempting to correct its causes and by the discreet use of soporifics. If the lessened sleep of advancing years does not cause weariness and irritability, the patient should learn to accept the

shorter hours as normal and adjust his living to this new nocturnal habit. When such sleep is insufficient for the individual, soporifics may be given: phenobarbital 1½ gr, or some other barbiturate, or chloral hydrate 10 to 15 gr. It is best to avoid the constant use of these drugs and to give them only when they are really needed.

## SMOKING

In the later years of life, as in youth, smoking does less

able Ten cigarets or two or three cigars daily should be sufficient Tobacco is harmful when smoking immediately gives rise to such symptoms as precordial pain, palpitation, dizziness and digestive disturbances In such cases smoking should be prohibited Patients with peripheral vascular disease must eliminate smoking completely, because there is no doubt that tobacco exercises a harmful effect on the progress of the disease The bronchitis accompanying emphysema is perpetuated and aggravated by smoking With these exceptions, elderly persons may be allowed to smoke in moderation As a matter of fact, older persons usually voluntarily cut down their tobacco consumption, because they begin to lose the taste for smoking or have persuaded themselves that it gives rise to some discomfort or hope that by such abstention they may prolong their lives

tongue, with absent papillae, may reflect a lack of the pellagra-preventive factor, nicotinic acid. In mild cases the tip and lateral margins of the tongue are reddened and swollen. In more severe cases there are intense glossitis, stomatitis and gingivitis. The tongue is red, swollen and smooth. There may be secondary infection with Vincent's organisms. Petechiae or hemorrhages into the skin in adults rarely point to vitamin C deficiency, in the presence of jaundice or serious impairment of intestinal absorption they may indicate lack of vitamin K. Rarely, a pellagra-like eruption may reveal a serious nutritional disorder.

*Skin and mucous membranes*—The skin should be inspected for its elasticity and dryness. The presence of senile keratoses should be observed particularly in sites where continuous irritation may give rise to malignant metaplasia. The toenails in old people are often thick and irregular and may lead to excoriations of the skin which favor infection. Large calluses on the soles of the feet may cause distress. Varicose veins should always be sought for. The state of the teeth should be studied from the point of view of infection, apical abscesses and pyorrhea, as well as in regard to their efficiency as chewing organs.

*Eyes*—Conjunctivitis is common, presbyopia almost universal, and glaucoma and cataract occur often enough to be kept in mind. The eyegrounds should be examined to discover the condition of the arteries and to detect possible hemorrhages or exudates which may point to serious hypertension or diabetes.

*Ears*—Impairment of hearing, especially for high tones increases with advancing years. Today this can be compensated for by appropriate electric hearing devices.

*Lungs*—Pulmonary emphysema is common in older people. It may cause dyspnea and cyanosis in the absence of heart

disease, and predisposes to recurrent attacks of bronchitis. The emphysematous chest is usually characterized by elevation and rigidity of the ribs, with calcification of the costal cartilages and a gentle kyphosis of the spinal column. A chronic cough and equivocal lung signs should not without further investigation be accepted as evidence of chronic bronchitis. Indolent pulmonary tuberculosis occurs with sufficient frequency in older persons to make it necessary to search for it in every case. Lung tumors should always be thought of when cough and chest pain develop for the first time after the age of 50. Physical signs may be scanty and confusing. The most common signs are those of atelectasis.

*Lymph nodes*—Enlarged lymph nodes in old persons are often evidence of tumor metastasis. Nodes in the left supraclavicular fossa may indicate cancer of the stomach, supraclavicular nodes may represent extensions from the mediastinum or lungs, and axillary nodes from the breast or the lungs. Multiple lymph node enlargement may be the result of lymphatic leukemia or lymphosarcoma.

*Heart*—The symptomatology is of more importance than are the physical findings in determining the integrity and the reserve power of the heart. Since coronary artery sclerosis is the most frequent cause of cardiac disorders in the aged, inquiry into symptoms of angina pectoris takes first place. The physician should further question in regard to dyspnea, orthopnea and cough. Headaches and vertigo in a hypertensive may point to the danger of a cerebral vascular accident. The pulse should be felt, not alone in the radial arteries but also in the femoral, posterior tibial and dorsalis pedis arteries, for circulatory impairment in the extremities is common. In doubtful cases oscillometric readings should be made of the arterial pulsations in the legs. The discovery of arteriosclerosis in the peripheral arteries does not justify the



inference that the visceral and especially the coronary or cerebral arteries are similarly affected, for the distribution of arteriosclerosis in the body follows no uniform pattern. Of course, the routine physical examination of the heart should be carried out. An electrocardiogram should be taken of every person over 50 who comes for a health inventory or who has symptoms that are at all suggestive of cardiac impairment. Often this will reveal an unsuspected myocardial lesion. The blood pressure should always be measured.

*Gastro intestinal tract*—Here too the symptomatology is more important than the physical examination. If one waits for palpable tumors to appear, it is too late. A story of newly developed indigestion or epigastric discomfort, especially when associated with loss of weight, should always arouse the suspicion of gastric carcinoma, particularly in men. A sudden change in bowel function, either constipation or intermittent diarrhea, calls for investigation of the colon for possible neoplasm. Roentgen examination of the stomach and colon, as well as gastroscopy or sigmoidoscopy, should be employed promptly when there is the slightest question of malignancy in the gastro-intestinal tract. Chemical test of the stool for blood or determination of the sedimentation rate of the red blood cells may offer valuable confirmatory evidence. Regulation of the bowels and control of gas is a major problem in older patients. A rectal examination should never be omitted. It may reveal a latent rectal carcinoma or an enlarged prostate. In the presence of hemorrhoids, never assume that bleeding is due to the hemorrhoids alone. There may be another, more serious, lesion as well.

*Genito urinary system*—The commonest disorders of this organ system in the aged are prostatic enlargement with difficulty in micturition in men and cystocele with faulty bladder control and cystitis in women. In a man with evi

dence of obstruction of urinary outflow the volume of residual urine should be measured in order to determine the degree of obstruction and the type of treatment that is indicated. Renal insufficiency due to nephritis or contracted kidneys is not so common. The most valuable test of renal function is the determination of specific gravity of the urine. A low fixed specific gravity is the best indication of kidney insufficiency, and unless this is present, blood nitrogen studies are unnecessary. The urine should be tested for sugar.

*Skeletal system and joints*—Disproportion between the weight of the body and the weight-carrying capacity of the feet often leads to static disturbances in the joints of the lower extremities. A heavy, pendulous abdomen associated with a lumbar lordosis causes back pain. Heavy pendulous breasts may cause higher dorsal pain. Postural defects, loss of muscle tone and obesity are responsible for many of the joint pains of older persons. Osteo arthritis of the spine is common in older people, and the degree of change found by roentgen examination does not correspond to the symptoms that may arise from disorders of the spine. Patients with extensive hypertrophic changes may have no symptoms, and those with minimal visible lesions may have great disablement. Infectious or rheumatoid arthritis occurs, but is rare, in elderly individuals. It is most unusual to relieve such a condition by discovery and removal of a focus of infection. The physician should always keep in mind that gout may be responsible for arthritic symptoms in older persons. A careful history may disclose ancient typical gouty attacks, and inspection may reveal tophi in the ears or gouty deposits in the olecranon bursa, or along the tendons of the fingers. Obscure bone pains should always suggest metastatic neoplastic deposits in the bones. Roentgen study and determination of sedimentation rate and serum phosphatase are then indicated.

*Central nervous system*—Tremor, as the first evidence of Parkinson's disease, is the symptom most commonly encountered. In the early stages there may be nothing but a slight tremulousness of the thumb. Absent knee jerks and pupils fixed to light should direct a search for cerebrospinal syphilis, but these signs may occur in diabetics or severe arteriosclerotics in the absence of syphilis.

*Blood*—A hemoglobin determination with an instrument that gives accurate readings is indispensable, because the color of older persons is highly deceptive. Secondary anemia may be the first intimation of a serious systemic disease or of inadequate diet. Polycythemia most commonly is secondary to advanced pulmonary emphysema. The sedimentation rate of the red blood cells is a useful test. An accelerated rate gives evidence of serious organic disease that must be uncovered.

*Fever*—The patient's temperature should always be taken by rectum. The mouth temperature in the aged may give a deceptively low reading, either because of slowed circulation in the mouth or because the patient cannot keep his mouth closed. The skin of old persons with fever often does not feel very warm, because of the lessened vasomotor response in the cutaneous blood vessels. Commonly, old people show less of a rise in temperature than do the young, and bronchopneumonia may run its course with no or very little febrile reaction.

*Pain*—It is claimed that elderly individuals experience pain less readily than do the young. In my experience this has not been the rule, except in severely debilitated persons with dulled sensoriums. Within one week I saw three patients aged 72, 77 and 85 suffering from severe pain of acute cholecystitis, from acute anginal pain accompanying coronary thrombosis and from intense pain in the wrists resulting from acute infectious arthritis respectively.

*Some principles of diagnosis*—The discovery of a lesion,

particularly in an older person, does not necessarily mean that that lesion is responsible for the symptoms of which the patient complains. Gall stones occur in over 10 per cent of women over the age of 45, and their discovery by roentgen examination is often accidental. The physician must establish a logical connection between such a lesion and the symptomatology, before ascribing the disease condition to that lesion. I have seen neoplasms of the stomach and colon overlooked because the patient happened to have gall stones. Similarly, dyspnea in an older person with hypertension and some cardiac enlargement should not automatically be ascribed to heart failure, it may be caused by emphysema or some other pulmonary process. A rapid pulse rate in such a patient may be due to hyperthyroidism, it does not always connote heart failure.

The unitary principle of diagnosis, the principle that one diagnosis must be found that will explain all of the lesions and symptoms, does not always apply in the case of chronic diseases in older persons. In these patients there is frequently a simultaneous derangement of several organ systems and a too rigid adherence to the principle of unitary diagnosis, which applies primarily to young people with acute diseases, may lead the physician astray. Men with coronary artery disease commonly suffer from peptic ulcer and occasionally from gastric carcinoma, hyperthyroidism not rarely is associated with organic heart disease, lung tumors are found in patients with pulmonary tuberculosis. In his diagnostic study of older persons, the physician should always keep in mind these possibilities and should not hesitate to seek multiple causes for symptoms that cannot be simply interpreted. Frequently two conditions—heart failure and pulmonary emphysema—contribute to such a common complaint as shortness of breath, and the relative importance of each of these factors

in causation of the symptom should be carefully evaluated.

*Treatment*—Some reference has already been made to the principle that, as far as possible the elderly patient's habitual routine of life should not be disturbed and that he should be helped and encouraged to remain a useful member of society. At times radical forms of treatment are necessary such as operations for cancers or prostatic obstruction, or complete bed rest for severe heart failure or amputation of a limb for gangrene but, by and large, the physician does best who treats his elderly patients with conservative methods.

*Bed rest*—The deleterious effects of bed rest on aged persons are well known. It induces loss of muscle tone with wasting of the muscles. There is a remarkable loss of calcium from the skeleton the calcium excretion may increase by as much as 400 per cent. The resulting calcinuria may be sufficient to cause formation of renal calculi. Bed rest further leads to urinary retention and aggravation of prostatism, constipation and loss of appetite. Pulmonary congestion and bronchopneumonia are common. These hazards have been excellently described by Laplace and Nicholson<sup>1</sup> who studied 30 patients with hip fractures. One half of those between the ages of 65 and 83 died. Careful studies revealed that these old patients begin to fail during the second, third and fourth weeks of immobilization. Because of their decreased muscle function and loss of muscle tonus there is peripheral blood stasis and blood accumulates in the venous capillaries and venules. As a result, the circulating blood volume is reduced, and peripheral vasoconstriction follows. These vascular reactions are similar to those that occur in shock. The resulting poor peripheral circulation causes local tissue anoxemia and

<sup>1</sup> Laplace, L. B. and Nicholson, J. T. "Prolonged Recumbency as a Contributory Cause of Death in Elderly Persons." *J. A. M. A.*, 110: 247 Jan. 22, 1938.

necrosis Decubitus ulcers are common, there is absorption of toxins from these infected areas The patient shows progressive apathy, toxic psychoses may develop and there is commonly a terminal bronchopneumonia Thrombosis of the veins of the pelvis or of the lower extremities occurs, and pulmonary embolism may result

If old people have to be kept in bed, great care should be taken to shift their position frequently and to have them move about in bed as much as possible Practically the only exception to this rule is in patients with severe heart failure Systematic muscular exercises and massage, particularly of the lower extremities must be carried out Especial attention should be paid to care of the skin Many old persons can sit up in a chair for at least part of the day Even walking to the bathroom or similar mild activities are beneficial Convalescence from bronchitis or bronchopneumonia, from operations or even from heart failure may be hastened by such partial mobilization In addition to activating the peripheral blood pumping action of the voluntary muscles, it promotes the pulmonary circulation by favoring excursions of the ribs and diaphragm

*Surgery*—Operation should not be withheld from anyone for reasons of age alone With proper selection of cases, with careful preoperative preparation and with meticulous operative technic, operations on elderly persons do not offer an undue risk to the patient When there is a true emergency, such as acute appendicitis, incarcerated or strangulated hernia or obstructive lesion of the bowel, there can be no debate, operation offers the only means to save life There are many disease conditions amenable to surgical correction that make life miserable, and the patient will gladly take a certain risk to gain relief from his discomfort Typical examples are prostatism, very large hernias, cataract and certain

forms of gallbladder disease. In each individual case the surgeon must weigh the several factors involved: the urgency of the situation from the point of view of saving life, the possibility of relieving serious discomfort and of prolonging life, the general physical condition of the patient, his mental attitude and the life expectancy of the patient as compared to the operative risk. Actual experience of many able surgeons has shown that the operative risk among the aged is not prohibitive and that many individuals have their lives prolonged and their existences made comfortable by well planned and executed surgical procedures.

At the Mayo Clinic<sup>2</sup> in the years 1939 and 1940, 1,364 operations were done on 1,204 patients aged 65 years or over, with an operative mortality of 9 per cent. In those aged 65 to 74 the mortality was 8 per cent, in those 75 to 79 it was 13 per cent. In those over 80 it was 35 per cent. In emergency operations such as those for repair of an incarcerated or strangulated hernia and acute appendicitis it ranged from 30 to 20 per cent and interventions to relieve intestinal obstruction had a mortality of 70 per cent. Major operation for benign disease showed a total mortality of 7 per cent, and for malignant conditions 8 per cent for operable lesions and 16 per cent for inoperable lesions. These are encouraging figures. They emphasize the gravity of emergency operations and suggest the advisability of correcting remediable defects earlier in life when it can be done with little risk. This applies particularly to hernias and to diseased gallbladders that give rise to persistent or recurrent symptoms.

The advisability of operation is determined in large measure by the general condition of the patient. Undernourished, dried out, pale individuals are poor operative risks, as are the

<sup>2</sup>Wilson, L. E. and Clagett O. T. "Surgical Procedures on Patients of Advanced Age," Proc. Staff Meet., Mayo Clin., 16:795, Dec. 10, 1941.

fat and flabby. In the case of the aged, an additional guide is his mental attitude—his will to live. Every candidate for operation should be subjected to a painstaking physical examination. An evaluation of the heart should determine the adequacy of the cardiac reserve and the degree of coronary artery sclerosis. The symptoms are as important as the physical findings. Dyspnea, cough, edema indicate a poor cardiac reserve. Angina pectoris points to serious involvement of the coronary arteries. The more readily the anginal seizure comes on, the poorer the surgical risk. Patients with coronary disease may develop cardiac infarction postoperatively. The unexpected mortality from this complication is about 5 per cent. Shock and hemorrhage are particularly prone to induce such an incident. Hypertension without serious cardiac complications does not add much to the hazards of operation. The size of the heart, as determined by fluoroscopy, is a useful guide. The larger the heart, the poorer the cardiac reserve. The quality of the heart sounds gives valuable information as to the state of the heart muscle. A dull, feeble first sound usually indicates myocardial impairment, a loud, forceful first sound suggests a strong heart muscle. An electrocardiogram should be taken routinely before operation. Extrasystoles are rarely of serious import. Auricular fibrillation or partial or complete heart block indicates cardiac impairment. The presence of demonstrable coronary artery disease, of calcareous aortic stenosis, of great cardiac enlargement, of auricular fibrillation or of symptoms or signs of heart failure gives evidence that the patient is a poor operative risk. In such cases operation should be undertaken only if there is a clear-cut indication and if the underlying disease will lead to still further cardiac damage or early death.

Pulmonary emphysema and fibrosis and chronic bronchitis favor the development of postoperative pneumonia. Such



pneumonias occur with particular frequency among the elderly. They can often be prevented by careful anesthesia and by the routine preoperative employment of sulfadiazine or of penicillin. The usual doses of one of these preparations should be administered for two days before the operation and continued postoperatively until every chance of pneumonia is past.

Kidney function should be tested in every case. A simple measure is the concentration test. If the specific gravity of the urine rises above 1.020 the kidney function is fairly adequate; if it remains between 1.015 and 1.020 there is impairment of renal function. Specific gravity that remains below 1.015 indicates serious renal damage. Determination of the urea nitrogen of the blood gives further information. In men the condition of the prostate gland should be investigated preoperatively, not alone because prostatic obstruction may have led to renal injury but because even patients with early prostatic hypertrophy may postoperatively experience difficulty in urination or transient complete retention of urine.

A complete blood count should be carried out preoperatively. Secondary anemia is common in the elderly.

In all but emergency cases every effort should be made to restore the patient to his optimum condition before operation is undertaken. If he is undernourished he should be fed carefully for several weeks. It is well to supplement the diet with liberal doses of vitamins, especially of the B complex. In the presence of anemia iron should be administered, and if this does not suffice to restore the hemoglobin resort should be had to transfusion. If there is heart failure the cardiac reserve should be built up with rest and digitalis. Similarly, the reserve power of the damaged kidney should be restored as far as possible. Even mild bronchitis is a contraindication to operation.

*Anesthesia*—The administration of anesthesia should be entrusted to a skilled anesthetist and adapted to the individual case. The routine preoperative use of morphine is unwise, it is too great a respiratory depressant. A dose of  $\frac{1}{8}$  gr. should rarely be exceeded. Ether is probably the safest anesthetic in the hands of the inexperienced. It is well tolerated by patients with heart disease. For those with pulmonary emphysema and chronic bronchitis it is too irritating to the air passages and should not be employed. In such cases cyclopropane is preferable. With this agent induction of anesthesia is rapid and smooth and recovery is rapid. A high concentration of oxygen can be maintained so that there is reduced danger of anoxia. With high concentrations of cyclopropane cardiac irregularities may occur. These should be avoided. If greater relaxation of the patient is needed curare may be administered intravenously while the patient is only lightly anesthetized with cyclopropane.<sup>3</sup> Spinal anesthesia is useful for operations on the prostate gland and on the lower extremities. A small dosage of the drug is usually sufficient, and this prevents the drop in blood pressure that accompanies the higher spinal anesthetics. Sudden lowering of blood pressure is hazardous to older patients, particularly to those with hypertension or with cardiac impairment. Intravenous anesthesia with sodium pentothal may be employed in selected cases but not for abdominal surgery or for operations lasting more than 45 minutes. This drug may cause respiratory depression in the aged so that the induction of anesthesia should be slow and the total dose kept small—not in excess of 1 to 1.5 Gm.

Postoperative complications that occur with particular frequency among elderly patients are bronchopneumonia,

<sup>3</sup>Knight R. T. and Baird J. W. "Anesthesia for the Aging and Aged." *Journal Lancet* 64: 183, 1944.

thrombosis of the peripheral veins and pulmonary embolism, cardiac infarction and psychoses. Early mobilization of the patient is one of the best means of preventing most of these untoward events. The position of the patient in bed should be frequently changed, and he should be compelled to flex and extend his legs at frequent intervals. If he is unable to move his legs massage should be given twice daily. He should be allowed out of bed at the earliest possible moment.

## *Chapter Five*

# **DISEASES OF THE CARDIOVASCULAR SYSTEM**

### **PREVALENCE**

DISEASES of the heart have become the leading cause of death with a death rate of 269 per 100 000. This rate includes persons of all ages. With each decade after 40 cardiovascular diseases loom ever greater as causes of illness and death. In the decade 40 to 49 heart diseases are responsible for about 25 per cent of all deaths. With each succeeding decade they exact a greater toll. Forty per cent of persons between the ages of 50 and 59, 52 per cent of those between 60 and 69, 62 per cent of those between 70 and 79, and 66 per cent of those between 80 and 89 die of a heart disease. Thus a knowledge of heart diseases is a major requirement for the physician treating persons of advanced years.

The phenomenal increased prevalence of heart diseases has led to the inference that some factors in our present civilization and way of life are responsible for it. It has been suggested that the hurry and strain of modern living and the increased urbanization and industrialization of our population bring about an early wearing out of our cardiovascular apparatus. Careful studies have demonstrated that this is not the case. The incidence of heart diseases in age groups under 40 has actually decreased due to the lessened incidence of

rheumatic fever and its sequel, rheumatic heart disease. In the older age groups the increase in frequency of heart diseases as a cause of death is of about the same magnitude as the decrease in infectious diseases (diseases such as pneumonia and tuberculosis) in these same age periods. Escaping infectious diseases, we live long enough to develop one of the heart diseases. Increased accuracy of diagnosis, particularly in the field of coronary artery disease, and certain changing fashions in diagnosis contribute to the apparent increased frequency of heart diseases. Thus the diagnosis "senility" has largely been eliminated and replaced by diagnosis of a cardiovascular condition. The increasing prevalence of heart diseases is due to the fact that we are living longer and are less subject to the infectious diseases. It reflects, in fact, favorable influences in the health of the people.

#### AGING OF THE HEART AND ARTERIES

The concept is erroneous that diseases of the heart and arteries at ages past 50 are inevitable manifestations of the aging process—that they are unavoidable and incurable.<sup>1</sup> The changes in structure and function of the heart and arteries that come with age are few and simple and do not give rise to clinical syndromes of disease, nor do they lead directly to death. The important senescent changes in the heart are pigmentation of the heart muscle fibers, atrophy of the heart muscle, enlargement of the valvular ostia and stretching and loss of elasticity of the valves. The electrocardiogram of the aged has no characteristics that distinguish it from that of younger persons. There is an increasing tendency to left axis deviation, an increase in the relative duration of systole and

<sup>1</sup> Boas, E. P. "Aging of the Cardiovascular System." *Bull. New York Acad. Med.*, 16: 607, October, 1940.

a lessened frequency of sinus irregularity. Prolongation of the PR interval is common. Studies have shown that about one quarter of persons over the age of 70, who are presumably normal, have electrocardiograms indicative of myocardial damage. These abnormal electrocardiograms, however, are evidence of arteriosclerotic myocardial disease, not of aging.

The arteries and veins become elongated and dilated from the progressive deterioration of their elastic tissue, and there is some thickening of the intima. The aorta serves as an example of the behavior of the large elastic arteries. As it grows older the aorta experiences a slow, progressive loss of elasticity. This process begins in the third decade and becomes accelerated after age 50. It is due to changes in the elastic properties of the elastic fibers in the media which lose their coil-like undulations and fail to re-coil when they are stretched.<sup>2</sup> In addition, microscopic studies show an increasing fine calcification of the media, of equal degree in both sexes, that develops rapidly after the age of 20 and is universal after age 50. The intensity of the calcium deposit is a function of the age of the aorta.<sup>3</sup> In youth the area of its cross-section, in the contracted state, is relatively small, but it can be expanded to three or four times its resting size. With advancing years the cross sectional area becomes progressively greater, with ever less capacity for distention.<sup>4</sup> This change in size and elasticity of the aorta bears no relation to the presence of arteriosclerosis.

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<sup>2</sup> Wilens, S. L. "The Postmortem Elasticity of the Adult Human Aorta: Its Relation to Age and to the Distribution of Intimal Atheromas," *Am J Path*, 13: 811, 1937.

<sup>3</sup> Blumenthal, H. T., *et al*. "Calcification of the Media of the Human Aorta and Its Relation to Intimal Arteriosclerosis, Aging and Disease," *Am J Path*, 20: 665, 1944.

<sup>4</sup> Winternitz, M. C., *et al*. *The Biology of Arteriosclerosis* (Springfield, Ill: Charles C. Thomas, Publisher, 1938).

Simple intimal thickening is evidently a physiological process, for it begins in the first years of life and occurs regularly in all but the smallest muscular arteries. Thickening of the intima after the fourth decade is due to increase of collagenous tissue, and shows fatty, hyaline and calcific changes as well. It gives rise to the typical picture of arteriosclerosis.

The maximum blood flow through the coronary arterial tree is about 35 per cent less after the age of 60 than in youth<sup>5</sup>

Changes in function of the heart and arteries occur with advancing years. The pulse rate remains fairly constant until about age 65 when it tends to increase to a slight degree. The cardiac output per square meter of body surface, measured under basal conditions, declines very slightly in old age. This decline results largely from the lessened oxygen consumption of the body.

The idea that with increasing age there is a progressive rise in blood pressure still finds general acceptance. The term hypertension is employed far too loosely. True arterial hypertension which so commonly leads to cardiovascular disease, is characterized by an elevation of both systolic and diastolic blood pressures. Systolic hypertension, without rise in diastolic pressure, has an altogether different mechanism and is the result, not the cause, of cardiovascular disturbances. The common form of hypertension met with in the aged is a systolic hypertension. In the later decades of life the systolic blood pressure may rise to about 140 or 160 mm of mercury, while the diastolic pressure remains unaltered.<sup>6</sup> This systolic hypertension and increase in pulse pressure is not caused by a narrowing of the peripheral arterial bed and does not place

<sup>5</sup> See J. W. C. ... "hypertrophy"

<sup>6</sup> ... te J Med  
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an added strain on the heart and arteries, it is the result of the loss of elasticity and the increased atherosclerosis and rigidity of the aorta and large arteries. The increasing length and width of the aorta and large arteries compensate for their loss of elasticity and help to keep the internal tension of the aortic wall constant. The increase in systolic pressure in the aged is an expression of loss of arterial elasticity, and in its effect on cardiovascular dynamics is a beneficent reaction.

A hyperactive carotid sinus reflex, manifested by temporary cardiac standstill and fall in arterial tension following pressure over the common carotid artery at its bifurcation, is more frequent in advanced age especially in men. The hyperactive reflex is commonly encountered in patients with arteriosclerosis of the carotid or of the coronary arteries. It may be the cause of syncope.

Although the functions of the cardiovascular system undergo relatively slight changes with advancing years, the circulatory system is not as competent as it was in youth. The aged make less demand on their hearts, they eat less, their physical activity is greatly lessened, they avoid extremes of cold and heat, and they are unaware that their hearts have become weaker, for they have little need for the cardiac reserves that they had in their youth. Under basal conditions the functioning of the heart and arteries suffices to meet the needs of the body, but the circulatory system, like the rest of the aging organism, has lost its resiliency and the power of quick adaptation to changing external stimuli. When outward circumstances compel aging individuals to take the buffetings of life as they did when they were younger, they discover that they lack adaptability, they cannot respond to the stresses of a physically active life.

In order of their importance, the conditions responsible for diseases of the heart and arteries in older persons are



arteriosclerosis    arterial hypertension    syphilis    rheumatic val-  
vular disease    hyperthyroidism

### ARTERIOSCLEROSIS

Arteriosclerosis may involve any of the arteries of the body. It gives rise to clinical symptoms of disease when it seriously impairs the blood supply of certain vital organs in particular the heart the brain the kidneys or the lower extremities. The cause of arteriosclerosis is unknown. Certain observations suggest that dietary and metabolic factors may be important in its genesis but knowledge is insufficient to permit prevention. Smoking physical and emotional strain cannot be regarded as causes of arteriosclerosis. Although the earliest evidences of arteriosclerosis are encountered on the autopsy table in youth clinical evidences of arteriosclerosis are uncommon before the age of 40. Their frequency increases up to about age 70. After this age the incidence seems to be lower for persons who survive the seventh decade apparently are born with stronger and more resistant blood vessels. Hereditary influences are commonly observed in the occurrence of arteriosclerosis. More than one member of a family is usually afflicted, and in certain families every member succumbs to arterial disease when he arrives at a certain age. Arteriosclerosis is about four times as common in men as in women. Diabetes favors the development of arteriosclerosis, so that diabetic women suffer from arteriosclerotic manifestations as frequently as do diabetic men.

*Pathology* — Arteriosclerosis is an all inclusive term applied to several distinct forms of arterial disease. One should differentiate calcification of the media or the Monckeberg form of arteriosclerosis occurring chiefly in the arteries of the extremities arteriolosclerosis of the small arterioles of certain

viscera of patients who have been subject to long standing hypertension and atherosclerosis which is the common form of lipid infiltration of the intima with atheroma formation seen classically in the aorta and larger arteries

The Monckeberg type of arteriosclerosis involves the muscular arteries particularly those of the extremities and occurs predominantly in males who are past middle life. It is most common in the femoral artery between Poupert's ligament and the popliteal fossa. The anterior and posterior tibial arteries are frequently involved, the radial artery less often. The calcium deposits begin in the interstitial substance between the muscle fibers but soon coalesce to form plaques and bands that encircle the artery. In the fully developed form the vessel feels like a goose trachea. In later stages bone formation may occur in the calcified areas. Calcification of the media does not lead to intimal thickening or to narrowing of the arterial lumen. It therefore does not give rise to symptoms of disease. It is easily visualized by roentgen examination.

Arteriolosclerosis represents the reaction of the small arterioles to the strain of a long lasting hypertension. It occurs chiefly in the arteries of the kidney, pancreas, spleen and liver. It is characterized by a subendothelial hyaline deposit and degeneration which gradually narrow the lumen of the vessel. There may be fatty infiltration of the hyaline deposits and secondarily there is increase in connective tissue cells and collagen. The muscle of the media atrophies as the intima becomes thicker. In the larger arterioles there is hyperplasia of the internal elastic membrane. As a result of the progressive narrowing and closure of the arterioles of the kidney glomeruli and tubules become atrophic and fibrous and the end result is the granular contracted kidney.

Atherosclerosis manifests itself at first as delicate yellowish flecks in the intima caused by deposits of cholesterol esters in

the interstitial substance and cells of the intima. It occurs in the aorta in children and in the anterior descending branch of the left coronary artery in many young adults. Gradually this fine granular lipid deposit increases, and accompanying it there are swelling of the cement substance and development of collagenous material in the subendothelial layer. Thus a localized swelling or plaque of the intima is formed. There follows a connective tissue reaction which some believe is caused by the cholesterol deposits, and layer upon layer of connective tissue may overlie the area of infiltration. This new tissue may undergo subsequent lipid change. Eventually the deeper areas of lipid infiltration undergo atheromatous degeneration, and free cholesterol crystals are deposited in the arterial tissues. Calcification may follow, or the atheromatous softened area may ulcerate into the lumen of the artery. Thrombi may form on the ulcerated surface. As a result of these complex changes the lumen of the artery may be greatly narrowed or completely obliterated. Hemorrhages commonly occur within the arterial wall about an atheromatous area, and such a hematoma of the artery wall may further narrow the lumen.

It is believed that the primary factor in atherosclerosis is a molecular change of the intima with imbibition of blood plasma and a deposit in the intima of cholesterol esters from the blood plasma. Certain mechanical factors play a rôle in determining the site of these arterial changes, for they occur in those portions of arteries exposed to stress or in regions where fixation of the vessel impairs its expansile mobility. Thus the earliest lesions in the aorta occur near the origin of the intercostal vessels, the abdominal aorta which is fixed against the spine is more involved than the thoracic portion, patches of arteriosclerosis occur chiefly on the posterior wall of the aorta and by predilection at the sites opposite the upper

and lower borders of the bodies of the vertebrae Moschowitz<sup>7</sup> has emphasized again the rôle of intra arterial tension in the genesis of arteriosclerosis and its development in the pulmonary artery when the tension of the lesser circulation is increased Winternitz<sup>8</sup> has stressed the fact that the distribution of the vasa vasorum is related to the occurrence of arteriosclerotic plaques

All the evidence suggests that arteriosclerosis is a disease, not a degenerative process of senescence If arteriosclerosis were a phenomenon of senescence, it should be encountered with some degree of uniformity in arteries throughout the body, it should appear with some measure of regularity and with similar frequencies among persons of different races and in the two sexes Experience shows that this is not the fact Arteriosclerosis, and particularly coronary sclerosis is far less common among Negroes than among whites It is very rare among the natives of northern China That arteriosclerosis occurs less often in women than in men is known to all clinicians and pathologists In women with hypertension, coronary sclerosis is frequently encountered Sclerosis of the pulmonary arteries is far less common than sclerosis of the systemic arteries, and when it is well developed there is almost invariably some process such as mitral stenosis or extensive pulmonary disease, that has led to increased pressure in the pulmonary circulation In diabetics the percentage mortality from arteriosclerosis is one third greater than for the population as a whole, and women are affected as frequently as are men In children widespread advanced arteriosclerosis is encountered in the presence of severe

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<sup>7</sup> Moschowitz E *Vascular Sclerosis* (New York City Oxford University Press 1942)

<sup>8</sup> Winternitz M C Thomas R M and LeCompte P M *The Biology of Arteriosclerosis* (Springfield Ill Charles C Thomas Publisher 1938)

nephrosis In hereditary xanthomatosis a condition characterized by xanthomatous nodules and a high blood cholesterol level arteriosclerosis is the rule and sudden death from coronary occlusion common

These observations clearly indicate that arteriosclerosis is not a simple wearing out of the arterial coats that comes with age but that it is a disease a disease it is true that manifests itself mainly but by no means exclusively during the period of senescence It remains a challenge to scientific investigation and a problem for constructive therapy

#### HEART DISEASE DUE TO SCLEROSIS OF THE CORONARY ARTERIES

*Pathology*—The degree of sclerosis may vary from a slight fatty deposit in the intima to extensive involvement of the vessel wall with calcification ulceration and obliteration of the lumen The sclerosis may be diffuse affecting most of the larger and smaller branches and giving rise to pipe-stem arteries or it may be localized An atherosclerotic plaque less than 1 cm in length may completely close the lumen although the rest of the artery may appear healthy Slow closure of an artery from arteriosclerotic narrowing favors the development of a collateral circulation so that when the affected vessel is finally obliterated by a thrombus no infarction of the myocardium may take place The development of collaterals is the rule in patients with coronary artery sclerosis and explains how they survive successive closures of different branches of the coronary arterial tree Hemorrhages are often found in the wall of a coronary artery around atherosclerotic areas The resulting swelling of the arterial wall may cause occlusion of the vessel or the hemorrhage may provoke the formation of a thrombus



FIG 2—*Above* thrombosis of a coronary artery *Below* section of the myocardium through a recent infarct The infarct appears yellow streaked with red



which will occlude the lumen at the site of the hemorrhage

When a thrombus forms suddenly in a large coronary artery which has not been narrowed for a length of time sufficient to allow the development of collateral channels, infarction of the myocardium takes place. Infarcts are found most commonly in the anterior wall of the left ventricle near the apex, in the papillary muscles of the left chamber and in the upper posterior portion of the left ventricle. A recent infarct represents an area of necrosis of the heart muscle. It has a soft consistency and slowly, in the course of four to eight weeks, is replaced by scar tissue which eventually becomes firm. Over the endocardial aspect of the infarct mural thrombi commonly form, on the pericardial aspect a reactive pericarditis occurs. Emboli may be released from the mural thrombus and may find lodgment anywhere in the peripheral circulation, most frequently in the arteries of the brain, of the extremities or of the kidneys. At times rupture of the heart through the softened infarct leads to sudden death.

Aneurysm of the ventricle results when a large area of heart muscle has been destroyed and replaced by a relatively thin sheet of fibrous tissue. This area then bulges in systole and may gradually increase in size.

Coronary arteriosclerosis gives rise to disturbances of cardiac function when the impairment of blood flow to the myocardium is sufficient to cause anoxemia of the heart muscle. Commonly the coronary blood flow is adequate with the body at rest, but with exertion the diseased vessels cannot accommodate the needed greater flow of blood. When such inadequacy is transient, the patient experiences typical anginal pain, which passes away when, with rest, the call for a greater blood flow is passed. If the exertion is more severe, anginal pain may be prolonged, and when the disproportion between the blood flow and the blood need of the myocar-





FIG 3—Aneurysm of the left ventricle

dium is sufficiently great actual myocardial necrosis or infarct formation can take place even without coronary occlusion. This condition is referred to as coronary insufficiency. Spasm of the coronary arteries may contribute to insufficiency of a coronary circulation already impaired by arteriosclerosis. The most common cause of reflex coronary spasm is cold. This explains the instantaneous onset of angina pectoris in many patients when they step out of the house on a wintry day. Reflexes arising in the gastro intestinal tract or gallbladder may also induce coronary spasm. Emotion and anxiety are common causes. Thrombosis of one coronary artery induces spasm in other branches. These reflexes are mediated by the vagus nerve and can be minimized by the employment of full doses of atropine. When as a result of repeated coronary closures and advanced coronary sclerosis myocardial damage has been extensive heart failure may occur. Under such circumstances cardiac hypertrophy may develop even in the absence of hypertension.

#### ANGINA PECTORIS DUE TO CORONARY ARTERY SCLEROSIS

Angina pectoris is rarely encountered in patients below the age of 40. It becomes increasingly common in the fifth sixth and seventh decades but after age 70 is less in evidence. It occurs about four times as often in men as in women. In women it is commonly associated with diabetes and hypertension. It is a common complication among older diabetics. Angina pectoris is seen less frequently among Negroes than among whites.

*Symptoms*—The presenting symptom is a pressing constricting breath taking pain experienced under the sternum and brought on by some form of physical activity or emotional stress. The pain is of such intensity and the sense of

being unable to breathe so great that the patient is compelled to stand still until the distress wears off in a few minutes. The actual site where the pain is experienced varies greatly. The substernal location is the commonest but frequently it is felt just to the left of the sternum and commonly it radiates to the back of the left shoulder and down the inner aspect of the left arm. However the pain may be felt in the right chest anteriorly in the back between the shoulder blades or at times in the epigastrium. Radiation may be to the right arm or to the neck and jaw. In some persons pain is not a prominent feature these complain of a choking sensation and inability to breathe. The most characteristic features of the anginal attack are that it comes with effort induces immobility and is relieved by rest. Any pain above the waist that has these characteristics is suspect of being anginal in nature. In the anginal attack there is usually no change in the heart rate or in the blood pressure although at times there is a transient elevation in pressure. Anginal attacks are provoked more readily by walking after a meal or in cold windy weather or when the patient is hurrying under stress and tension. The anginal syndrome may come on insidiously at first appearing only under great effort. Gradually through the years the pain comes more often and on less provocation. In other cases the onset is sudden. A man who has been feeling perfectly well suddenly experiences his first anginal attack while walking or while shoveling snow or dancing. This may be quite severe and may last about 15 minutes. He recovers promptly but from that time on effort always produces anginal pain usually of less intensity than that experienced with the first attack. In these cases it is clear that the sudden onset must have been associated with some immediate permanent change in caliber of a coronary artery such as a hemorrhage into a coronary arterial wall. In other

cases anginal symptoms first appear after a frank coronary thrombosis

Gastro-intestinal symptoms are a common feature of the anginal attack. The patient complains of epigastric oppression or fullness and often finds that belching relieves both the abdominal and precordial distress. A hiatal hernia may cause symptoms simulating those of angina pectoris (see p. 305). There are interesting associations between angina pectoris and peptic ulcer.<sup>9</sup> The symptoms of peptic ulcer and anginal symptoms may develop suddenly and simultaneously, and occasionally acute peptic ulcer may be associated with coronary thrombosis. Attacks of angina pectoris may occur, at rest, two or three hours after meals and during the night at the hours characteristic of pain from ulcer. Epigastric localization of anginal pain may be conditioned by a preexisting peptic ulcer. When symptoms of angina pectoris and peptic ulcer coexist, successful treatment for the symptoms of ulcer may induce remission of the anginal syndrome. The common denominator of the two syndromes seems to be heightened excitability of the vagus mechanism. Symptoms of peptic ulcer may arise from vagal activity in the stomach, and symptoms of angina pectoris may result from vagal coronary vasoconstriction. A functional disturbance of one organ may reflexly, via the vagus nerve, induce a disorder in the other.

Physical examination is usually unrevealing. Hypertension occurs in only one half the cases. In the absence of hypertension the heart is not enlarged. Usually the heart sounds are of good quality. No murmurs are heard except in cases of calcareous aortic stenosis, which may also give rise to the anginal syndrome. Sclerosis of the peripheral arteries or of the arteries of the retina may or may not coexist. The pres-

<sup>9</sup> Levy, H. and Boas, E. P. Angina Pectoris and the Syndrome of Peptic Ulcer, *Arch. Int. Med.*, 71: 301, March, 1943.

ence of sclerosis in one artery does not permit the inference that other arteries in the body are similarly affected, for the intensity of the arteriosclerotic process in different parts of the body may be quite unequal. The electrocardiogram is often normal. Only when previous closure of a coronary artery with resultant cardiac damage has taken place are electrocardiographic changes encountered. The electrocardiograms may take any of the forms seen with myocardial disease.

*Diagnosis*—The diagnosis is based chiefly on the history, for in classic cases there may be no abnormal physical or electrocardiographic signs. So the symptomatology must be inquired into with great care. One should ascertain the mode of onset and duration of the pain, as well as its site, its character and radiation. Precordial pain is a common complaint. Benign precordial pain, which does not connote coronary disease, is felt about the apex, it is often sharp and sticking, or it is a dull, continuous distress. It is not provoked by exertion but often comes at rest, in bed or after a heavy day's work. It does not compel the subject to stop in his tracks. In women this type of pain is especially common, even when associated with hypertension it need not indicate coronary artery disease. Angina pectoris is rare in women under the age of 50, and in women up to the age of 60 it is unusual unless there is associated hypertension or diabetes. In older women it is not uncommon. Nerve root pains caused by spondylitis may be felt in the precordium. They occur at rest or with movement of the trunk. At times gall stone colic may cause confusion. The diagnosis of angina pectoris is confirmed by the history of a past coronary artery thrombosis or by the discovery of an abnormal electrocardiogram.

*Prognosis*—The prognosis for angina pectoris due to coronary artery disease is difficult to give for the individual case. By and large, it is much more hopeful than was thought 10

years ago. Many of these patients live for 10 to 20 years without significant change in their status. Yet there is an element of uncertainty in every case. Sudden death or coronary thrombosis may occur at any time. Whereas the patient should be given the hopeful point of view, some member of the family should be told that although things may go well for a long time, a serious complication may occur unexpectedly, the next day, the next week, in a month or in a year. The more readily the attacks come on and the more easily they are induced, the graver the prognosis. When there are many attacks a day and when attacks occur at rest, the outlook is much more serious, for in such cases a coronary occlusion may be expected at any time. These cases are discussed in further detail in the following section on coronary insufficiency.

#### MANAGEMENT

*Prevention*—Coronary artery disease cannot be recognized before it manifests itself by symptoms, except in rare instances when a routine electrocardiogram may disclose evidences of myocardial injury. Yet it is most desirable to recognize disease of the arteries of the heart at the earliest possible moment, for there is ample evidence that coronary artery thrombosis is often induced in such diseased vessels by unusual stress or strain. In men over the age of 40 any pain in the chest, neck, arms, jaws or epigastrium induced by physical activity should arouse suspicion and call for careful examination, including electrocardiography. The more carefully one enters upon the history of each individual case, the more often does one find symptoms that, in the light of subsequent events, can be recognized as those of angina pectoris. Or one discovers that a sudden effort or emotional strain was followed by an aggravation of symptoms or by a true coronary

thrombosis In such individuals the occurrence of coronary thrombosis can often be postponed if the diagnosis is made early and if appropriate measures are taken It is wise, too, in older men to take routine electrocardiograms before subjecting them to the strain of a major operation

*Treatment*—Although it is impossible to influence the progress of arteriosclerosis or to bring about a regression of lesions already existing treatment can give much relief and prolong life Therapeutic efforts should be centered on the patient rather than on the diseased heart or arteries Should the patient be told the true nature of his disorder? The terms "angina pectoris," and "hardening of the arteries of the heart" are so well known to the laity and strike such terror in their hearts that it is rarely wise to employ them The patient is too apt to recall the case of a friend or relative or some prominent individual who died suddenly from coronary artery disease Yet the patient should be informed that his heart is injured, for only with such knowledge can he take care of himself properly Whenever possible it is best to use some general expression such as heart strain, or to explain that the heart is slowly losing the elasticity and responsiveness of youth and that it must be spared If the patient guesses the truth, the physician should take pains to explain that many patients with coronary artery disease, even after a thrombosis, live long lives, and that with care they may look forward to many years of fruitful activity It is appalling how careless many physicians are in pronouncing diagnoses that, without qualification, terrify the patient Unfortunately many go still further and, either in their attempts at making the patient slow the tempo of his life or else for fear that they will be censured for overlooking a serious lesion in case the patient dies suddenly, inform him that he has a serious heart lesion and that at any time he

may fall dead Every year I see many individuals who have been so frightened by their physicians that the psychic disorganization provoked by their fears outweighs by far the symptoms of their disease It is essential to paint the picture as brightly as possible, while guiding the patient to realize that his future depends largely upon himself At the same time the physician should take some member of the family into his confidence and explain more fully the nature of the disease But here, too, emphasis should be placed on the uncertainty, rather than on the fatality of the condition Many may live for years, but death or serious invalidism may occur at any time

Treatment, as in all cases of heart disease, calls first for the regulation of the patient's activities and reference may be made to the principles laid down for the treatment of patients with essential hypertension In the case of sufferers from angina pectoris, there is a more certain guide to activity, and that is the anginal pain itself The patient must learn so to control and adjust his daily routine that it does not give rise to anginal attacks The appearance of pain is always an indication that the heart has been overtaxed If, by slowing the tempo of his activities, the patient can go through the day without anginal attacks, the routine of his life should not be interrupted There is no evidence that in the ordinary case the complete interruption of physical activity, with the adoption of a state of semi invalidism, serves to prolong life It certainly leads to hypochondriasis and psychic and moral deterioration This is observed most strikingly in patients who have sufficient disability insurance to enable them to live on the proceeds These unhappy individuals learn to live for their hearts alone, are always apprehensive and never at ease They are constantly searching for symptoms arising in the heart This state of mind is subconsciously furthered by the



knowledge that demonstrable disability is necessary for a continuance of the disability benefit

Whenever possible, therefore, the patient should be kept at his work. It may be necessary to curtail the hours of work or the intensity of his activities, but so long as he can go through the day without distress he is far better off busy than idle. Many physicians are afraid to allow their patients to work for fear that if they suffer a major seizure while at work, the doctor will be censured for permitting them too great activity. It is my conviction that aside from instances of definite acute overstrain, physical activity or inactivity bears no relationship to the progress of coronary artery disease, and that attacks of coronary thrombosis are just as apt to lay low the patient who is endeavoring to conserve his strength at home as one who has resumed his normal occupation. This point should be explained to the patient's family. In the case of men who are engaged in heavy manual occupations, change of work should be insisted on. Yet it is astonishing how many of them, through force of necessity, continue on their jobs for many years without serious distress. The physician should be guided by the reaction of the patient. As soon as it is evident that a daily routine or particular form of activity gives rise to symptoms, the disturbing factor should be eliminated. Slow, measured physical activity during a full working day does less harm than do sudden spurts of exertion. Lifting a heavy object, running to catch a bus, shoveling snow on a cold winter day, partaking of a heavy meal and intense emotional excitement often precipitate acute cardiac infarction. Such activities should never be undertaken.

Exercise for its own sake should be avoided, although short leisurely walks of a mile or so may be permitted. All games, as well as swimming, should be prohibited. Although many patients with angina pectoris can play golf without distress,

it is wiser to prohibit this activity too. Bathing, not swimming, may be allowed in the absence of surf, when the water and air are warm. Driving an automobile is usually well borne unless the patient is a nervous, tense driver. He should be warned again hurry and should try to avoid situations in which a sudden, severe pull on the wheel or other forcible action is necessary. Of course, he should not be allowed to change tires or endeavor to make any other heavy repairs. In the average case there is little danger that the patient will die suddenly at the wheel before he can bring the car to a stop.

There is no one diet that benefits all patients with coronary artery disease. Obesity calls for reduction in weight. The point of importance is to eat lightly, never to permit a sense of epigastric fullness after eating. At times it is better to take five small meals rather than the conventional three meals a day. Foodstuffs which the patient has learned by experience cause gas or epigastric distress should be avoided. Anginal attacks are provoked most readily by physical exertion on a full stomach. The patient should make it a habit to rest in an easy chair for one half hour to an hour after every meal. Many patients experience their only pain on leaving the house early in the morning after breakfast. They should have breakfast early enough so that they can relax and read the newspaper before leaving the house.

When angina pectoris occurs at rest, two or more hours after meals, it may be due to relative hypoglycemia. Harrison and Finks<sup>10</sup> have shown that although blood sugar values at such times may be only slightly subnormal, symptoms can often be relieved by the administration of sugar and reproduced by the injection of insulin. Patients with such peri-

<sup>10</sup> Harrison T. R. and Finks R. M. Glucose Deficiency as a Factor in the Production of Symptoms Referable to the Cardiovascular System. *Am Heart J.*, 26:147 August, 1943.

odicity of anginal attacks often find permanent relief from a diet poor in carbohydrate and high in protein with supplementary feedings between meals

Patients with the combined syndromes of peptic ulcer and angina pectoris should receive a modified Sippy diet with frequent feedings. In addition they should receive full doses of atropine, enough to induce dryness of the mouth, i.e., from 1/150 to 1/75 gr three or four times a day. One third to 1/2 gr of phenobarbital given three times a day lessens reflex excitability. In some cases alkalis or colloidal aluminum hydroxide may be useful.

Although anginal seizures are at times provoked by sexual intercourse, complete abstinence often has a worse effect upon the patient. He should be taught to avoid sexual excitement, but when he feels the need, he should have normal sexual relations.

Smoking in moderation may usually be permitted. There are certain few patients who can relate their cardiac pain directly to smoking. These must give up the use of tobacco completely, but the majority of patients with angina pectoris are not harmed by smoking 10 to 15 cigarets or a few cigars daily. Recent suggestions that coronary artery disease is determined by an allergic response to tobacco are as yet unproved.

Alcohol in moderation promotes relaxation and often exerts a beneficial effect in these patients.

*Medication*—Nitroglycerin is the one invaluable drug in the treatment of angina pectoris. It is purely symptomatic in its effect, relieving the pain of the acute attack, most probably by dilating the coronary arteries. It is of no value when given several times a day in the hope of preventing attacks. If certain activities such as defecation or the first walk on leaving the house in the morning regularly induce anginal seizures, nitroglycerin taken immediately beforehand may

prevent the onset of pain. The patient with angina pectoris should always carry with him tablets of nitroglycerin and take one promptly at the first signs of genuine precordial oppression. The tablet should be placed under the tongue and allowed to dissolve there. The usual dose is 1/100 gr which may be repeated if necessary as soon as the effects of the first dose have worn off. Some patients develop severe throbbing headaches from this dosage, and for them 1/150 gr may be sufficient. Amyl nitrite in the form of pearls that are crushed in a handkerchief, allowing the patient to inhale the volatilizing fluid, is equally efficacious but is more likely to cause headaches. Since we are seeking an immediate transient effect, these two preparations of the nitrites are the ones preferred. Sodium nitrite and erythrol tetranitrate are slower in their action and are more apt to be followed by severe headache. Given three or four times a day in ½ gr doses they are useful when anginal attacks are very frequent.

There still is controversy as to whether the purine derivatives such as aminophylline and theobromine relieve coronary spasm and improve the circulation of the heart. I have rarely, if ever, seen benefit from these drugs when given orally, and not infrequently they cause gastric disturbances. Theobromine sodium salicylate may be given in doses of 5 to 15 gr three times a day and aminophylline in a dosage of 1½ to 3 gr three times a day. There are many proprietary modifications of these purine derivatives that have no particular virtues over the two drugs that have been cited. These preparations usually contain a sedative as well and owe their effects largely to this. Large doses of aminophylline given in a manner that permits rapid absorption of the drug often do prevent recurrent anginal seizures. At least 7½ gr should be given intravenously, or preferably by rectum in the form of a suppository, or by instillation of the powder dissolved in

water The dose may be repeated several times a day Some patients do not tolerate the drug because of irritation of the rectum or the occurrence of nausea Various muscle and pancreatic extracts as well as testosterone propionate have been recommended under the supposition that they will promote dilatation of the coronary arteries These, too, I have found of little value

When anginal attacks are related to the onset of extra systolic irregularity, the abolition of the arrhythmia by the use of quinidine sulfate may bring about a striking increase in the capacity to work without pain The dosage of quinidine sulfate for this purpose is 3 gr three or four times a day

Potassium iodide is of no benefit

Next to nitroglycerin the most valuable drugs in the treatment of angina pectoris are the sedatives Phenobarbital in a dosage of  $\frac{1}{2}$  gr three times a day is probably the most widely used, although all of the barbiturates are equally effectual In some cases particularly when there is arterial hypertension, 5 gr of chloral hydrate and 10 gr of sodium bromide taken three times a day bring considerable relief When the attacks are chiefly nocturnal, the patient should take an early very light evening meal and a full dose of the sedative before going to sleep

*Surgery*—Operative treatment is indicated chiefly in patients who develop anginal attacks on the slightest effort and who do not improve under strict medical treatment The various forms of cervicothoracic sympathectomy are attended with an operative mortality of about 21 per cent although they at times give relief They have been largely abandoned and properly so Equally good results with much less risk can be achieved by injecting small quantities of 95 per cent alcohol paravertebrally into the upper four or five thoracic posterior ganglia This procedure interrupts sympathetic

pathways from the heart to the spinal cord. This method was first suggested by Mandl<sup>11</sup> and elaborated by Swetlow<sup>12</sup> and White<sup>13</sup>. Injection of the posterior ganglia is difficult and should be done by a surgeon skilled in the technic, who does paravertebral injections frequently. In many instances disturbing painful intercostal neuritis which may last several months, develops after the injection. If the injection has been skillful, the relief of anginal pain is immediate and usually permanent.

A systemic disturbance that burdens the heart with increased work may make manifest a latent coronary arteriosclerosis by provoking cardiac pain on slight exertion. Relieving the heart of this unnecessary strain may lead to complete disappearance of symptoms of angina pectoris at least for a time. Such a combination is found in hyperthyroidism and in severe anemias. In both conditions the heart labors to maintain an increased minute volume flow. The immediate indication is relief of the systemic disturbance. In patients with hyperthyroidism and angina pectoris, subtotal thyroidectomy is urgently indicated and is usually well borne. Many hesitate to operate, but there is no other recourse and delay usually leads to disaster. In severe anemias the heart compensates for the diminished oxygen carrying power of the blood by making it circulate faster. Restoration of the blood to normal relieves the heart of this extra work. In hyperchromic anemia treatment with liver extract brings speedy relief. The secondary anemias, encountered in patients with

<sup>11</sup> Mandl F. Weitere Erfahrungen mit der paravertebralen Injektion bei der Angina pectoris. *Wien klin Wochenschr* 38:759, 1925.

<sup>12</sup> Swetlow G. I. Angina Pectoris. Paravertebral Alcohol Block for Relief of Pain. *Am J Surg* 9:88, July 1930.

<sup>13</sup> White J. C. Angina Pectoris. Treatment by Paravertebral Alcohol Injection or Operation Based on Newer Concepts of Cardiac Innervation. *Am J Surg* 9:98, July 1930.

angina pectoris, that are amenable to treatment are usually due to continuous or repeated bleeding, as from hemorrhoids or fibromyomata. The bleeding must be stopped, by operation if necessary, and the blood picture restored by the administration of large doses of iron. At times preliminary transfusion is helpful. This can be done without danger if citrated blood is given slowly by intravenous drip, so that the flow can be regulated at will.

#### CORONARY INSUFFICIENCY

When anginal attacks occur many times during the day, and particularly when they occur repeatedly at rest or during sleep, the conclusion is warranted that there is advanced narrowing of one or more of the coronary arteries. Minimal increases in the circulatory requirements induce cardiac anoxemia because of this extreme interference with coronary blood flow. There exists a state of coronary insufficiency. In such patients one can predict that a coronary artery occlusion will occur in a short time. Rarely patients may continue with innumerable anginal attacks for many months before this event takes place, but more commonly it occurs much sooner. Physical signs and the electrocardiogram may remain normal until the closure actually takes place.

There is another form of coronary insufficiency in which the patient experiences a severe prolonged attack of anginal pain lasting from half an hour to an hour. This usually follows some unusual exertion or excitement. During the attack these cases are indistinguishable from actual coronary thrombosis, but the subsequent course shows that no myocardial infarction has taken place, because no fever, leukocytosis or permanent electrocardiographic changes develop during subsequent days. Within a few days the patient feels

as well as he did before the attack. At times actual myocardial infarction may take place under such circumstances, without closure of a coronary artery. This occurs particularly when the coronary flow is greatly slowed following shock or hemorrhage. This is the most extreme form of coronary insufficiency and cannot be distinguished from coronary thrombosis except on the autopsy table.

*Treatment*—Coronary insufficiency, with innumerable anginal attacks daily, is most difficult to treat. The patient should be completely immobilized in bed for many weeks. The aim in therapy is to spare the heart every unnecessary strain until a collateral circulation has developed of sufficient degree to take over the function of the vessel that is almost

closed as soon as the patient is able to get up and take from 20 to 30

tablets a day. In these cases sodium nitrite or erythrol tetranitrate, in doses of from  $\frac{1}{2}$  to 1 gr. four to six times a day, or  $7\frac{1}{2}$  gr. suppositories of aminophylline several times a day, may reduce the number of attacks. If attacks occur during sleep, a dose of erythrol tetranitrate should be taken just before retiring. Sedatives should be given freely. Often these measures do not halt the attacks, and recourse must be had to codeine and morphine. Papaverine in  $\frac{1}{2}$  gr. doses may be useful. These patients suffer so greatly that one is tempted to do almost anything to give them relief. Indeed they beg that some drastic measure be carried out. Yet in such cases it is very risky to proceed with paravertebral alcohol block. The shock of this operation may readily induce a coronary closure. The diet should be very light and very small in quantity. In these cases the prescription of an 800 calorie diet is indicated. It lightens the work of the heart during digestion and, if persisted in for four weeks, reduces the basal metabolic rate and so indirectly spares the heart. If under this



regimen attacks diminish in frequency and intensity, the patient may gradually be granted increasing activity. Far more often, even during strict treatment, a coronary occlusion occurs.

#### CARDIAC INFARCTION AND CORONARY THROMBOSIS

The clinical manifestations of coronary thrombosis are actually the result of cardiac infarction. Infarction is caused most often by a thrombus occluding a coronary artery, but it may be due to coronary occlusion from any cause—thrombosis, embolism, hemorrhage into the arterial wall or rupture into the lumen of an atheromatous abscess in the arterial wall. Infarction may result from prolonged coronary insufficiency without arterial occlusion, as may occur in shock or hemorrhage. Coronary thrombosis may occur without subsequent cardiac infarction if there is a rich collateral coronary arterial circulation. In such a case the patient may manifest the classic initial symptoms of coronary thrombosis, but recovers within 24 hours without developing fever, electrocardiographic changes or other evidences of myocardial infarction. The discussion of cardiac infarction in the following pages includes consideration of the syndrome of coronary thrombosis.

Cardiac infarction may occur at any time in a patient suffering from angina pectoris. It may also strike, without warning, a person who has had no previous symptoms of heart disease. Not infrequently certain prodromal symptoms may give warning of an impending attack some days before it actually occurs. In patients with angina pectoris, as has been pointed out, the occurrence of frequent attacks at rest indicates the imminence of a coronary closure. The sudden onset of anginal attacks at rest or an unusually prolonged attack after some effort usually indicates that a coronary closure is



FIG 4 -Infarct of the anterior aspect of the left ventricle Arrow indicates intraventricular thrombus

on the way Several days or a week may elapse during which there are repeated lesser attacks of pain without sequelae, until finally a major seizure indicates that infarction has taken place

Although many times a cardiac infarction may occur without evident cause, in a large proportion of cases the onset of symptoms can be traced to a definite unusual event in the patient's life <sup>11</sup> The most frequent cause is some severe bodily effort, such as pushing an automobile out of a ditch, or some exceptional strain while at work Physical exertion in cold weather is especially prone to cause attacks Every winter I see several men who develop their first symptoms while shoveling snow or while struggling through snow drifts Overeating and overdrinking at banquets and weddings, particularly when followed by dancing, is a common antecedent story In other cases drinking large quantities of iced drinks may precipitate an attack Sexual intercourse as well as unusual emotional excitement may be a direct cause of cardiac infarction Infections, such as influenza and pneumonia, and operations in patients with coronary disease are commonly followed by cardiac infarction

The clinical picture of a major cardiac infarction is well known The patient is seized with an agonizing constricting substernal pain, and feels as though the chest were being compressed by a heavy weight, the pain may radiate down one or both arms or up the neck He becomes tremendously apprehensive and breaks out in a cold sweat Symptoms of shock appear The blood pressure drops, the pulse becomes feeble and thready Some die in the acute attack, but in most cases there is slow recovery from the shock, particularly after morphine has been administered In some the pain subsides

<sup>11</sup> Boas E P "Some Immediate Causes of Cardiac Infarction" *Am Heart J*, 23 1, January, 1942

in the course of an hour, in others it may persist with gradual lessened intensity for 24 hours. With recovery from shock the patient feels fairly well but tremendously weak from the terrible experience he has been through. His appearance, if first examined at this time, may be very deceptive. His color is good, the pulse slow, the heart sounds of good quality, and the blood pressure back at its former level. After 12 to 24 hours myocardial necrosis begins. Absorption of autolytic products of the softening infarct provokes a constitutional reaction and the damage to the heart muscle becomes apparent. The temperature rises. It may range from 101 to 104 F and gradually recedes in from five to seven days. There is leukocytosis which may reach between 12,000 and 20,000. At the same time the pulse becomes rapid, the first heart sound becomes dull, and if damage has been severe there may be gallop rhythm with accentuation of the second pulmonic sound. In about 10 per cent of cases a pericardial friction rub becomes audible. This friction rub is very inconstant and may come and go within a few hours. Rarely it persists for several weeks. The blood pressure drops. Normal blood pressure may be reduced to 90/60, and in a patient with previous hypertension, figures almost as low may be observed. In severe cases congestive heart failure becomes manifest, chiefly by dyspnea and moist râles in the lower lobes of the lungs. In the severest cases there may be early pulmonary edema. Cardiac irregularities may occur. Most common are frequent extrasystoles. There may be auricular fibrillation. With massive infarction of the posterior aspect of the left ventricle there may be partial or complete heart block. If the patient survives, these irregularities disappear within a few days. The electrocardiogram usually gives evidence of cardiac infarction within a few hours of its occurrence. The two classic pictures are those of anterior infarc-

tion with a high take-off of the T wave in leads 1 and 4, which changes in the next few days and shows Q waves and negative T waves in leads 1 and 4, and those of posterior infarction in which initially there is a high take off of the T wave in leads 2 and 3, followed by the development of Q waves and negative T waves in leads 2 and 3

Death from ventricular fibrillation or rupture of the ventricle may occur at any time during the first weeks after a myocardial infarction. If the patient improves, there is gradual recession of all of the signs and symptoms. However, the first heart sound may remain dull, and the blood pressure may remain low. In cases with severe myocardial damage, evidences of heart failure may persist. Between the fifth and the fourteenth day after infarction, embolism may occur from a thrombus that has formed on the endocardial surface of the left ventricle. It may cause a cerebral vascular accident, most often hemiplegia, it may cause a renal or splenic infarct, or the embolus may lodge in one of the arteries of the extremities. A large embolus may be arrested at the bifurcation of the aorta, giving rise to the clinical picture of the so called saddle thrombus.

Commonly myocardial infarction is attended with far fewer symptoms. There may be no shock, the pain may be of only half an hour's duration, and the constitutional reaction may be slight and transient. A patient may experience a true cardiac infarction and return to work after a night's sleep or, at most, a few days' rest in bed. Many patients are sent by their physicians to my office for examination within one to seven days after having experienced a cardiac infarction. Although many survive such careless handling, not a few develop congestive failure, because the injured heart has been overtaxed by this inadvisable physical activity. I have seen not a few unnecessary deaths in persons who have pursued

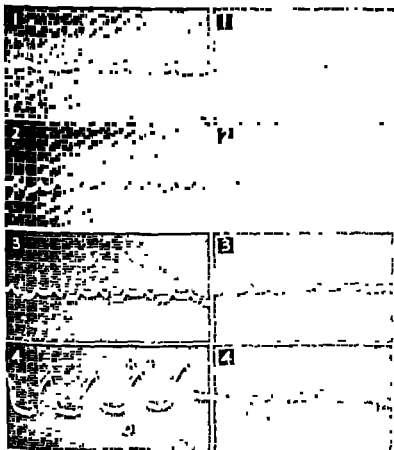


FIG 5—Electrocardiograms *Left* acute infarction of the anterior aspect of the left ventricle note elevation of ST segments and Q waves in leads 1 and 4 *Right* healed infarct of the anterior aspect of the left ventricle Note Q waves and negative T waves in leads 1 and 4

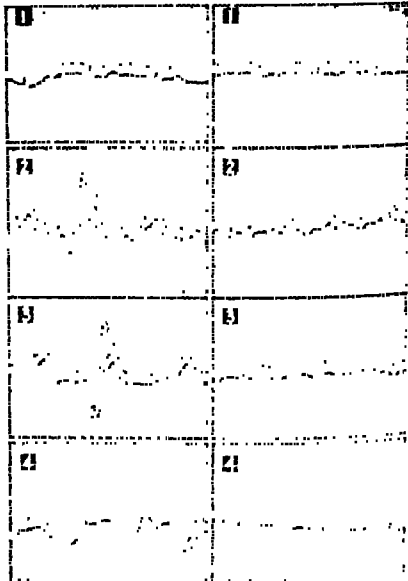


FIG. 8—Posterior infarction of left ventricle. *Left*, acute infarction (4 hours old) of posterior aspect of left ventricle, note elevation of ST segments and Q waves in leads 2 and 3. *Right*, healed infarct of posterior aspect of left ventricle (same case). Note Q waves and negative T waves in leads 2 and 3.

their normal activities following an unrecognized infarction

The mistaken diagnosis of simple angina pectoris is often made in these cases. Anginal pain lasting more than half an hour is almost always followed by cardiac infarction. In every such case the physician should assume that an infarction has taken place until it has been disproved. Careful recording of the temperature, determination of the white blood cell count and the sedimentation rate of the red blood cells will usually lead to a correct interpretation of the clinical picture. An electrocardiogram should be taken for positive findings will make the diagnosis certain. A normal electrocardiogram, however, does not exclude the possibility of cardiac infarction.

*Treatment*—Major cardiac infarctions call for prompt and rigorous treatment. The patient is in agonizing pain and in shock. Large doses of morphine should be given hypodermically. It is useless to commence with as small a dose as  $\frac{1}{4}$  gr. One half gram should be given at once, and if there is no measurable relief within 15 minutes or so, another  $\frac{1}{4}$  or even  $\frac{1}{2}$  gr. should be administered without further delay. To lessen reflex spasm in the unaffected coronary arteries atropine sulfate  $1/75$  gr. should be given with each dose of morphine. The patient should be put to bed at once. He should not be allowed to walk or to help himself in any way, but should be carefully carried. Any exertion no matter how slight, may cause sudden death or precipitate serious heart failure. If the patient is very ill, it is best not to move him more than is necessary, but to make him comfortable wherever he may happen to be. The body should not be warmed. The cold skin is due to peripheral vasoconstriction, a protective device to maintain blood flow through the vital organs.

Restlessness calls for more morphine or a sedative such as sodium amytal, 3 gr., or chloral hydrate 10 to 15 gr. The



tion. Usually after 1 gr. of morphine the pain is controlled and the patient is fairly comfortable. When morphine causes vomiting it should be used sparingly, or dilaudid 1/30 gr. may be used instead. The shock gradually passes off in one to 12 hours. During the first 24 hours following cardiac infarction the patient is suffering from peripheral vascular failure rather than from heart failure. Therefore cardiac stimulants should not be given until and unless heart failure supervenes.

During the first 24 hours no solid food should be given and only enough fluids to prevent discomfort. No attempt should be made to induce bowel movement. The patient may not void at all or at most very little. This too calls for no interference. Because of the state of shock there is actual partial suppression of urine and the kidneys do not resume their normal function for several days.

At the end of 24 hours, often sooner, the acute stage characterized by shock has passed. The patient has regained a good color, the pulse is full and the blood pressure has risen in part, from its original low level. Even the heart sounds may have a rather strong quality. If the patient has been too ill to be moved to an appropriate place, this is the best time to transport him to his home or to a hospital on a stretcher and in an ambulance. Then follows the stage of reaction to the cardiac infarct. The temperature rises, the pulse again becomes weak and rapid, congestive râles appear at the bases of the lungs and the liver may become engorged. With this the heart sounds become feeble and a pericardial friction murmur may be heard. Some pain may persist, but it is rarely severe.

Pain can be controlled with codeine. Half a grain may be

given every four to six hours as necessary. Morphine should be used if necessary. In addition,  $\frac{1}{2}$  gr of phenobarbital three times a day helps to promote quiet and relaxation. The patient should be kept at complete rest on his back for at least four weeks. He should be fed, bathed and turned in bed, he should not be allowed to do anything for himself. The fluid intake should be restricted to about 1 000 cc a day, food to about 1,000 calories. The diet should be simple and light.

No attempt should be made to induce a bowel movement during the first three or four days. The effort of defecation or of an enema may overtax the heart. If there should be much distention, a rectal tip should be inserted, but only in case of absolute necessity should a small enema be given.

If congestive failure appears, or if the pulse is rapid and there is gallop rhythm, treatment should be directed to the heart. In the absence of such signs no specific cardiac medication is necessary. Heart failure, no matter whether it occurs immediately or after several days, always calls for the administration of digitalis. The fear that digitalis may do harm in the presence of an acute infarct, that it may cause rupture of the heart or induce ventricular extrasystoles or tachycardia is unfounded. If heart failure is severe the patient should be quickly digitalized. Four cat units should be given intravenously, and at the same time 4 more units by mouth. Six hours later 4 more cat units may be given by mouth, and 12 hours later another 2 units. After this it is best not to exceed a dosage of 2 cat units a day. This dosage may be continued for a week and then reduced to 1 cat unit daily. The best preparation of digitalis is digitoxin, which is commercially available as Purodigin or Digitaline Nativelle in 0.2 mg tablets. The initial dose is 5 tablets, or 1 mg, and this is followed by 1 tablet, or 0.2 mg, once a day. Given in these amounts, digitalis can do no harm and is almost always

helpful in combating heart failure. Some prefer to use strophanthin intravenously for the initial doses. One quarter of a milligram may be given as the initial intravenous dose, and this may be repeated in six hours. After this, medication should be continued with digitalis by mouth. If, in spite of this medication, much moisture remains in the lungs and the liver remains swollen, 2 cc. of one of the mercurial diuretics should be given intramuscularly. In the presence of severe heart failure, intravenous administration of these preparations may induce pulmonary edema. The dose may be repeated in three days, if necessary. When these measures fail, the situation is quite desperate and further medication does not offer much promise. Four grains of aminophylline in 10 cc. of distilled water may be given intravenously, or caffeine sodium benzoate may be given hypodermically in doses of  $7\frac{1}{2}$  gr. every three hours. Adrenalin is not useful in these cases and may cause serious harm.

Aminophylline by mouth serves no useful purpose and may upset the patient's stomach. Some employ glucose intravenously, in the hope of improving the nutrition of the heart muscle. I have found it of no value. In fact, such intravenous medication, by increasing the blood volume, places an extra strain on the heart and may do harm.

If congestive failure is severe and there is much cyanosis and dyspnea, the administration of oxygen will ease the burden of the laboring heart and improve the nutrition of the myocardium by supplying it with well oxygenated blood. Oxygen is best administered through the B. L. B. mask. If the patient cannot tolerate the mask the oxygen tent may be used, and if other methods fail the simple nasal catheter can be employed.

Sudden death following cardiac infarction may result from ventricular fibrillation. Some authors have advocated the

routine use of quinidine sulfate in doses of 3 gr three or four times a day to prevent this eventuality. It seems unnecessary to administer this drug in every case, but when extrasystoles appear, particularly at rapid heart rates, it should be given until uninterrupted sinus rhythm has re-established itself. Auricular fibrillation following cardiac infarction rarely calls for quinidine, for as a rule the rhythm spontaneously reverts to normal in a few days. It is better in such cases to employ digitalis to slow the ventricular rate.

Two of the most serious complications of cardiac infarction are embolism from a mural thrombus overlying the infarct and pulmonary embolism arising from phlebothrombosis in the legs. If the formation of such thrombi could be prevented by lessening the coagulability of the blood, such embolism might be prevented. Dicumarol has been administered to patients with cardiac infarction in the hope of achieving this result. Dicumarol retards thrombus formation by de-

creasing the coagulability of the blood. This time should be kept at about 20 per cent of normal. The initial dose of 300 mg is given as soon as the diagnosis is made. About two days are required for the drug to manifest its action. Subsequent doses are given daily after the prothrombin time is ascertained. The average daily dose is 75 mg, but if the prothrombin time approaches normal the dose should be increased, and if it is depressed below 40 per cent the dose should be omitted until the level is higher. It is too early to evaluate the usefulness of this drug. Any bleeding tendency is a contraindication to its use.

The sedimentation rate of the red blood cells is a good indicator of whether or not healing of the infarct is complete. When the sedimentation rate has become normal, one may

assume that the soft infarct has been fully replaced by scar tissue and that it is safe to mobilize the patient. No matter how well the patient appears to be doing, he should be kept on his back in bed for a full month. If at the end of this period there are still evidences of congestive heart failure, if the pulse is rapid and there is gallop rhythm, absolute rest must be continued. If the cardiac function is good, he may be allowed to sit up in bed and within a few days may spend a few hours a day in a chair. Gradually his range of activity may be increased so that at the end of another four weeks he is walking freely about the house. He should, however, avoid all undue strain and take several rest periods during the day. At the expiration of two months from the time of the coronary thrombosis, complete healing of the myocardium has taken place and cardiac function has been largely restored. The patient may be allowed to leave the house and gradually test out his capacity for physical activity. The range of freedom that may be permitted is determined by the patient's response to effort. The appearance of anginal pain or dyspnea on exertion calls for limitation of activity along the principles laid down in the discussion of angina pectoris.

The interpretation of the patient's response to effort after he has experienced a coronary artery occlusion is often difficult. He is usually fearful, always on the lookout for some sign of cardiac embarrassment. He walks and handles himself as though he were some fragile object that might break if used too roughly. It is a slow and painstaking task to teach him the balance between sensible caution and irrational timidity. The evaluation of his symptoms, too, may lead to error. Sticking precordial pain, unrelated to effort, is common during convalescence from a cardiac infarction. It may be caused by the pull of pericardial adhesions that form so commonly, it has no clinical significance and should be

ignored Muscular weakness and ready fatigue often have marked psychic components and may call for the prescription of greater activity There is such danger that these convalescents from a serious cardiac accident may become confirmed neurasthenics that the physician should devote special effort to dissipate their fears and to help them learn the degree of activity in which they may indulge with safety

Many of these patients, particularly those who have experienced their first attack of coronary thrombosis, recover fully and have no sequelae and no disturbing symptoms They should be encouraged to resume their former occupation unless it is one calling for much bodily labor It is doubtful whether complete retirement and a life devoted to guarding the heart against possible stresses and accidents actually accomplishes its end Recurrent coronary thromboses seem to come in those who have reduced their living to a mere existence as frequently as in those who have resumed the usual course of life It is true of course, that unusual strain should be sedulously avoided A sudden momentary effort may do more harm than a year's tempered activity and may, in fact, induce a thrombosis in a coronary artery The patient should learn not to push himself to avoid competition to escape the drive to which we so often subject ourselves He should give heed to warnings of fatigue and of anginal pain, no matter how slight He should arrange his work so that he can carry on with comfort With these simple precautions, the majority of those who have recovered from a cardiac infarction can resume the interrupted thread of their lives

There are others in whom anginal pain on greater or less effort develops or persists after the coronary thrombosis These should be handled according to the principles laid down for the treatment of angina pectoris Still others, but

these are few, have continuing symptoms and signs of congestive heart failure after the coronary closure. The treatment of these is outlined in the following section.

*Prognosis*—About 80 per cent of patients recover from their first cardiac infarction. The outlook becomes more serious with each subsequent attack. The presence or absence of hypertension does not seem to affect the outlook, but marked cardiac enlargement is an unfavorable element. It favors the development of heart failure. The older the patient, the more serious the prognosis. The mortality from cardiac infarction becomes greater as each successive decade of life has passed. In the individual attack, the early onset of heart failure is an unfavorable sign. The severity of the heart failure, as well as of the constitutional reaction, is also an indicator of the seriousness of the individual attack.

#### MYOCARDIAL FIBROSIS WITH HEART FAILURE

When there have been repeated cardiac infarctions or when the coronary arteries have undergone slow but wide spread occlusion, diffuse myocardial fibrosis results. In time so much of the myocardium is destroyed and replaced by fibrous tissue that the contractile power of the remainder is insufficient to maintain the circulation. There may be an aneurysm of the left ventricle. Aneurysm of the left ventricle is suggested by a diffuse, forceful, visible and palpable apex impulse accompanied by a very faint first heart sound. The diagnosis is confirmed by fluoroscopy which reveals a protrusion of the left cardiac border which bulges in systole while the rest of the ventricle contracts. In simple myocardial fibrosis the heart is usually enlarged and the first heart sound is faint. When the heart begins to fail, the pulse becomes rapid and the pulmonic second sound becomes accentuated.

The failure is primarily of the left ventricle, so that dyspnea and congestion of the lungs are the leading symptoms. Commonly attacks of paroxysmal dyspnea, or so called cardiac asthma, form part of the clinical picture. These attacks usually occur at night, awakening the patient from sleep. They may be quite mild and may be relieved when he sits up for a short time. More often they are quite intense, with agonizing dyspnea and cold sweat. In the severest attacks, pulmonary edema occurs and may be fatal. Gallop rhythm is a constant sign in this form of heart failure and pulsus alternans is frequently encountered. When left ventricular failure is far advanced, the right ventricle fails too, the liver becomes congested and enlarged, the neck veins become full and edema of the legs may develop. Anginal pain is unusual after heart failure sets in.

In spite of the advanced state of the cardiac lesion, treatment of these patients often gives great relief and prolongs life. Complete bed rest is essential. If the patient has not been taking digitalis the drug should be given in the dosage outlined in the section on cardiac infarction. If the patient has been taking digitalis, maintenance doses only should be given. It is unwise to give more than 1 cat unit daily when dosage is to be continued for a long time. The dose may be doubled, if believed necessary for a week or so at a time.

Successful treatment concerns itself primarily with the water metabolism and effects excretion through the kidneys of the excess water in the body. Even in the absence of visible edema this may amount to 10 lb or so. The presence of this fluid, which is associated with an increased blood volume, embarrasses the heart. After it has been removed, the feeble heart is able to maintain the circulation, but only so long as the water balance of the body is maintained. As soon as fluid again accumulates in the tissues, cardiac distress returns.



The importance of maintaining the fluid balance in the body was known to the Ancients Celsus wrote

It is wise to measure the circumference of the abdomen with a string daily On the following day one must always note whether the abdomen has become larger or smaller because only if the body is becoming thinner are the medicines working It is also wise to measure the fluid intake and the urine of the patient because there is hope for recovery only when the patient excretes more fluid than he takes in <sup>15</sup>

He also advised incision of the skin above the ankles for dropsy and abdominal paracentesis for ascites

Successful treatment hinges on a careful recording of the intake and output of fluid In addition to the water ingested in the form of fluid drinks, soup and ice, a large amount is taken with solid food Vegetables, fruit, meat in fact most foodstuffs contain large quantities of water Water is excreted through the kidneys, through the lungs, through the skin, as visible and invisible perspiration, and through the intestines with the stool Fortunately the amount of water ingested as solid food approximately equals that excreted by the lungs, skin and intestines, so that for practical clinical purposes it suffices to measure the actual fluid intake and the urinary output These should be measured for every 24 hour period and recorded daily A glance at these figures shows at once whether or not the patient is making any progress The urinary output should always exceed the fluid intake by a few hundred cubic centimeters When the patient is not too ill to stand, a better check is obtained by weighing him daily at the same hour Daily fluctuations in weight are due to variations in water retention With these records as guides the physician is prepared to initiate treatment

The fluid intake should be limited to 1,000 cc in 24 hours

<sup>15</sup> Celsus Aurelius Cornelius *De Medicina* B III chap XXI

The Karell diet—800 cc of milk and nothing else in 24 hours—is useful, but the need for such rigorous measures has largely disappeared with the introduction of the mercurial diuretics. The salt intake should be minimal, but it is rarely necessary to prescribe a strict salt free diet. A diet containing only 1 Gm of salt daily will induce diuresis and permit the patient to drink larger quantities of water. All cooking and baking, even of bread, must be without salt. Such a diet is difficult to prepare and many patients refuse to adhere to it. It is very useful when dehydration cannot be achieved by other methods.

*Diuretics*—Ammonium chloride should be given in a dosage of 20 to 30 gr three or four times a day. It is conveniently administered in the form of the 7½ gr enteric coated tablet, which is well tolerated by the stomach. When ammonium chloride is absorbed the ammonia is synthesized into urea, and the liberated chlorine ion produces acidosis. It is this acidosis which is responsible for the diuresis. If the heart failure and edema are not too great, these simple measures may suffice to rid the body of its excess fluid and to restore the circulatory balance. In the presence of markedly impaired renal function these acid producing diuretics should be used with caution, for they may bring about serious acidosis. This may be suspected if the breathing becomes rapid and deep, and can be verified by measuring the carbon dioxide combining power of the blood.

If the edema is massive or if dyspnea is distressing diuresis can be hastened by the administration of one of the mercurial diuretics. These act upon the kidney tubules, preventing the resorption of water that has been excreted by the glomeruli. The best of the mercurials are mercupurin, mercurhydri and salyrgan. They may be given intravenously or intramuscularly in a dose of 1 to 2 cc. If the patient has veins that

are readily accessible for injection, the intravenous method is best, for it is painless and the action of the drug is more rapid. Great care should be taken to avoid introducing any of the drug into the tissues around the vein, for this causes a painful induration that may slough and take weeks to heal. When intravenous technic is too difficult, it is better to give an intramuscular injection although this may be painful. These drugs may be given in suppository form, in which they are almost as efficacious as when given by injection. However, many patients cannot tolerate the suppositories, for they may cause severe rectal irritation. In patients with chronic water retention the oral administration of these diuretics, one tablet three times a day, may maintain adequate diuresis and render injections unnecessary. Some patients experience abdominal cramps and diarrhea from the oral dosage. The dose of the mercurial may be repeated every three to seven days, depending on the degree of water retention. Even in the absence of gross edema, the active diuresis provoked by these drugs often abolishes dyspnea and leads to restoration of circulatory equilibrium.

In old people, particularly, diuresis caused by administration of a mercurial drug may be followed by intense weakness or by muscle cramps. These drugs bring about a great elimination of salt, as well as of water, and it is the excessive salt loss that is responsible for these symptoms. They may be relieved by the administration of salt by mouth. The occurrence of such symptoms calls for smaller subsequent dosage of the mercurial. Usually a dosage can be found that will be tolerated.

In the absence of advanced kidney disease the mercurial diuretics are quite safe. Albuminuria alone is no contraindication to their use, for it may be due to passive congestion of the kidneys. But when the urine contains many red blood

cells and casts it is unsafe to employ these drugs, unless it can be established that there is little kidney damage or no active renal disease. When the kidney has lost its concentrating power, when the specific gravity of the urine is persistently below 1.012, they should be used with great caution. Active dehydration under such circumstances may lead to azotemia and to death from uremia. I have observed this sequence on a number of occasions.

One of the best diuretics for patients with persistent severe heart failure due to advanced myocardial disease is urea. It has two major disadvantages—its very disagreeable taste, and the fact that it often provokes intense thirst. The dose is 1 oz two or three times a day. It is best administered in orange juice or beer. Under its steady use, a urine output of from 2,000 to 4,000 cc. can easily be maintained. Urea is cheap, it can be bought for less than a dollar a pound; it is non-toxic. I have observed many patients who have taken it continuously for several years with uniformly good results. While urea is being taken, the blood urea nitrogen is usually high; it may amount to 50 or 60 mg. per cent, but this causes no ill effects and is no indication for stopping the drug. Once a patient has become reconciled to the taste of the drug and has learned how, by its continuous use, he can control his dyspnea as well as the distress occasioned by a congested liver, he is unwilling to give it up. The maintenance dose should be the smallest one that will keep him in water balance. The mercurial diuretics may be given to supplement the action of urea, but this is rarely necessary except in advanced states of dropsy.

Compared to the powerful diuretics that have just been discussed, drugs such as theobromine and aminophylline are relatively inactive. I rarely find occasion for their use. Aminophylline may be given in a dosage of 1½ to 3 gr. three

times a day, and theobromine sodium salicylate 10 to 15 gr three times a day. Their diuretic effect is often short lived.

Some patients recover from their severe heart failure and maintain their circulation without the need for further diuresis. But there are many who again retain water as soon as the diuresis is stopped. They remain chronic invalids. Their exercise tolerance is minimal; they can walk but little. Most of them are confined to the house, many are bed ridden. But as long as diuresis is maintained they are comfortable provided they do not overtax their physical reserves. The ultimate prognosis of course is poor. Few survive more than a year or two.

Success in treating these patients depends on eliminating every ounce of excess water from their systems and then keeping them "bone dry." This can best be achieved by watching their weight and noting the minimum weight that can be attained at which they are comfortable. The patient should weigh himself every morning on arising and record the figures. A sudden increase of 3 to 5 lb. always indicates water retention and warns of impending symptoms of left ventricular failure. The immediate administration of a diuretic will eliminate this surplus fluid and again restore the balance of the circulation. Such gain in weight usually follows excess physical activity or the ingestion of too much fluid. These factors too, should be kept under constant regulation. With careful supervision, using the weight as a guide, these patients do remarkably well. But the briefest relaxation of vigilance and care results in recurrence of heart failure.

Attacks of *paroxysmal dyspnea* call for emergency treatment. Morphine sulfate, at least  $\frac{1}{4}$  gr. should be given hypodermically at the onset of the attack. If there is associated pulmonary edema that does not at once respond to the opiate and if the veins of the neck and arms are full, phlebotomy

of 500 cc may be done. Usually, unless there is associated right heart failure, there is no indication for phlebotomy, indeed, the abstraction of blood is difficult because the veins are empty. The intravenous administration of 8 gr aminophylline calcium in 10 cc of water may abort the attack. If the patient has not been taking digitalis,  $\frac{1}{4}$  mg of strophanthin or 2 to 4 cat units of an appropriate digitalis preparation should be given intravenously. This should be followed by rapid complete digitalization.

At times the application of tourniquets to the four extremities will serve to diminish the circulating blood volume and act as effectually as a phlebotomy. The tourniquets should be applied so as to arrest the venous return but still permit transmission of the pulse wave. They may be left on for a number of hours. On releasing them, care should be taken to do so gradually and to free one limb at a time else the rush of blood that has been pooled in the extremities may overtax the heart. Hot water bags and hot blankets should be applied to give warmth to the body for the temperature is usually low. Aside from morphine, digitalis and aminophylline, the best drug is caffeine sodium benzoate  $7\frac{1}{2}$  gr which may be given hypodermically every hour or two. As an emergency stimulant, coramine is at times of value. It is only effective when given intravenously, when it acts as a powerful stimulant to respiration and circulation. Its effect, however, is fleeting.

As soon as the patient has recovered from the acute episode of paroxysmal dyspnea active diuresis according to the methods outlined above should be instituted. This will often serve to prevent further attacks. Since the introduction of the mercurial diuretics, thoracentesis and abdominal paracentesis are rarely necessary, but if dyspnea is intense and apparently caused by the accumulation of fluid in the serous cavities, withdrawal of this fluid may give immediate relief.

*Diabetes mellitus* is commonly encountered in patients with coronary artery disease. The principles of treatment are the same as in uncomplicated diabetes, with one exception. Too rigorous dieting with a view to maintaining a normal blood sugar concentration or too liberal use of insulin may precipitate arterial thrombosis in the coronary or cerebral arteries or in sclerotic arteries of the extremities. The diet should be adjusted to maintain the patient's normal weight and to keep the urine practically sugar-free. If this can be achieved without insulin, all the better. If insulin is used, care should be taken to avoid doses that produce hypoglycemia, for a too sudden or too radical reduction in the blood sugar is often accompanied by attacks of angina pectoris or even of coronary thrombosis. Most patients with both diabetes and coronary artery disease are past middle life, and the diabetes is relatively mild. They often feel better when they excrete a little sugar in the urine than when they are under a too rigid regimen.

*Periarthritis of the shoulder*, giving rise to pain in the left shoulder and arm, is seen so often in patients with coronary artery disease that the association seems to be more than a coincidence. Often the acute inflammation about the shoulder joint occurs within a few days after a cardiac infarction, or it may develop weeks later. When precordial pain radiates to the left shoulder and arm, the left shoulder is apt to be the one involved; when radiation is to the right, the right shoulder is affected. Thus in patients with coronary artery disease we see chiefly periarthritis of the left shoulder, in contradistinction to the more common right-sided lesion. Otherwise the clinical picture does not differ from that in patients with normal hearts.

Periarthritis of the shoulder is a loose term that includes several conditions. One form is associated with calcific de-

posits in the supraspinatus tendon. The other common form leads to a "frozen shoulder" and is due to tenosynovitis of the long head of the biceps and its sheath.<sup>16</sup> It may begin with a mild ache in the shoulder which is exaggerated by external rotation. The pain and limitation of motion gradually increase, and there may be a secondary scalenus spasm that induces pain in the neck and back of the shoulder. There is marked limitation of abduction and external rotation. Motion may be so restricted that fibrous ankylosis is simulated. The full picture may develop quickly. The pain is persistent and severe, it is exaggerated by movement, and lying in bed may be impossible because no weight can be borne on the shoulder. Trophic disturbances may appear in the hand, and there may be marked atrophy of the shoulder girdle. The condition is self-limited and gradually after many months clears up completely.

Anginal pain often radiates to the shoulder and arm, it may be chiefly manifest in this location. When such a periarthritis occurs in a patient with angina pectoris, the shoulder pain is apt to be severe, apparently because the sensory nervous pathways of this region have already been sensitized by the radiation of the pain arising in the heart. It is often difficult to determine whether such shoulder pain arises from disease in the heart or in the shoulder. If the pain comes on effort and if it is associated with typical substernal pain, it is anginal in nature. If the pain is continuous and aggravated by movements of the arm, if lying or turning in bed provokes severe pain in the shoulder and if on examination there are local tenderness and limitation of abduction and external rotation, the pain is not a manifestation of angina pectoris but is due to disease of the structures around the shoulder.

<sup>16</sup>Lippman R. K. "Frozen Shoulder Periarthritis Bicipital Tenosynovitis," *Arch. Surg.*, 47: 283, September, 1943.



joint. The close association as well as the differentiation of these two conditions are emphasized because they are so often confused. Thus, a patient whose heart calls for little treatment is confined to bed or greatly restricted and is erroneously given nitroglycerin for the relief of arthritis.

When periarthritis of the shoulder is acute, absolute immobilization and the application of wet dressings for a few days are essential. Heat is often comforting, but it may aggravate the pain which frequently is sufficiently severe to require morphine or codeine. Aspirin and aminopyrine often give relief. In the earlier stages roentgen therapy may be efficacious. As the acuteness of the condition subsides gentle massage and diathermy treatments are beneficial. It may take weeks or months for the pain fully to subside. At times a maneuver devised by Dr. E. Libman gives instantaneous relief. With the thumb, pressure is exerted in the anterior triangle of the neck over the brachial plexus until a spot is found where such pressure reproduces the shoulder pain. At this point strong pressure, often causing severe pain, is exerted for one minute. Not infrequently this causes immediate and permanent relief from the shoulder pain. The mechanism of this legerdemain is not understood. In some cases this procedure is ineffective, and in others pain and disability return within a day or two. Injection of 20 cc. of 1 per cent procaine solution into the muscles about the shoulder at the sites of spots that are tender to pressure may give quick relief.

#### ARTERIOSCLEROSIS OF THE CEREBRAL ARTERIES

Arteriosclerosis of the cerebral arteries affects the brain much as arteriosclerosis of the coronary arteries affects the myocardium. Interference with the blood supply to a portion of the brain causes local anoxemia and loss of function of the

affected area. If the anoxemia is very transient, no permanent damage ensues, but if it is at all prolonged the affected nerve tissues die and an irreversible lesion results. As in coronary disease, occlusion of a cerebral artery may be due to progressive atherosclerotic narrowing; it may be caused by thrombosis superimposed upon the arteriosclerosis; or there may be hemorrhage into the vessel wall and into the brain, due at times to rupture of military aneurysms; or there may be embolism. Shock and hemorrhage, by causing marked prolonged insufficiency of the cerebral circulation, may induce cerebral softening without thrombosis of one of the arteries of the brain. Cerebral arteriosclerosis and resulting cerebral vascular accidents are by no means confined to patients with hypertension. As in the case of coronary artery disease, hypertension is found in about one half of the cases. Since the symptoms in the hypertensive patients are much like those in patients with normal blood pressure they will be discussed together. There has been much dispute whether or not spasm of the cerebral vessels can occur and whether transient seizures of hemiplegia or aphasia are caused by such momentary spasm or by small hemorrhages that are quickly absorbed. Probably both conditions occur.

Just as attacks of angina pectoris may give warning of impending coronary thrombosis, so fleeting disturbances of cerebral function may be manifest some time before the cerebral vascular insult occurs. Headaches, sudden vertigo and confusional states are not uncommon. There may be sudden aphasia or merely an impediment of speech which quickly disappears. There may be transient hemiplegia. Sooner or later a major attack occurs. The symptoms depend on the size of the lesion. In the case of hemorrhage, the additional factor of increased intracerebral pressure contributes to the clinical picture. In the classic severe case there is

sudden loss of consciousness accompanied by incontinence of urine and feces. Coma may develop more slowly during a period of 12 hours and be heralded by intense unilateral headache vomiting and increasing mental dulness or confusion. Breathing is slow and stertorous and may be Cheyne Stokes in character. In fatal cases consciousness does not return breathing becomes more and more labored and rapid and the temperature rises steadily and inexorably often reaching 107 to 108 F just before death. The pulse is little accelerated and is strong the blood pressure is often elevated. Evidence of paralysis of one side of the body can be elicited by close observation of the patient. For the first day or two the deep reflexes are abolished but subsequently they become hyperactive on the paralyzed side and the Babinski sign becomes apparent.

In favorable cases consciousness gradually returns usually within 24 hours but I have seen patients recover completely after five days of unconsciousness. Within 12 to 24 hours the temperature rises and a leukocytosis occurs. Fever lasts from three to seven days and is due to absorption of autolyzed products from the infarcted area in the brain. At times the cerebral injury may cause a disturbance of temperature regulation. Full appraisal of the extent of the paralysis can be made a few days after the apoplectic seizure. Recovery is slow. Power first returns to the lower extremity beginning in the muscles of the hip and thigh followed by those of the leg. The arm is last to recover and often remains useless even when the patient can walk quite well. Under appropriate treatment, function in the paralyzed limbs may continue to improve for several months. In case of aphasia speech may return promptly or the aphasia may persist in its full extent or only slightly. Alexia difficulty in reading is a distressing complaint that often accompanies aphasia.

Unconsciousness occurs in only half the cases. Often hemiplegia, monoplegia or aphasia occurs without disturbance of the sensorium. The onset may be sudden or slow. In the case of hemorrhage, the lesion usually develops rapidly, although it may progress in the course of the first 24 to 48 hours. The symptoms of cerebral embolism appear suddenly and dramatically. In older persons, the source of the embolus is almost always a mural thrombus of the left ventricle following coronary thrombosis. Rarely, it is derived from a vegetation of subacute bacterial endocarditis. Thrombosis of a cerebral artery is, as a rule, slow in development, and the resultant symptoms develop gradually in the course of 24 to 48 hours.

Changes in the mental capacity and emotional pattern of the patient are variable. In many, the intellectual powers are unimpaired and the patient may resume his normal life without any evidences of deterioration. One of the most striking examples of such recovery is Louis Pasteur, who lived 28 years after having experienced a stroke and who, in those years, accomplished some of his best work. In many, however, there results a deterioration of mental capacity. Often there is emotional instability, particularly when the lesion involves the thalamus. There are episodes of uncontrollable laughter or crying. After repeated or extensive cerebral vascular insults, mental deterioration becomes marked, giving rise eventually to the picture of arteriosclerotic dementia.

Not every cerebral vascular insult gives rise to the classic

no major episodes with visible neurological sequelae, but rather repeated small apoplexies, the nature of which is

often unrecognized <sup>16a</sup> There may be fainting or acute vertigo, or a sudden severe headache with vomiting The symptoms pass off within a few days and are attributed to indigestion Or the patient may suffer a so called nervous breakdown with extreme weakness and exhaustion Following such attacks he may never again be his former self, he may become irritable, forgetful and exhibit marked changes in character or temperament, become emotional, untidy or suffer from insomnia The true nature of the illness is commonly overlooked because of the rapid physical recovery after such an episode, and because of the absence of motor or sensory disturbances The best diagnostic lead is a painstaking history which will reveal that the patient's illness dates from a sudden unexpected seizure, such as fainting, vertigo or vomiting the time of onset of which can be fixed precisely Such minor cerebral vascular injuries may begin in the fifth decade, and there may be years of well-being before the occurrence of a second one Sooner or later such apoplectic strokes recur and lead to multiple areas of encephalomalacia, with resultant physical and mental deterioration and eventual gross neurologic defects Ten or 20 years may elapse before a major seizure causes death

*Treatment*—The prevention of cerebral vascular accidents is rarely possible, except in certain patients with hypertension When the blood pressure, particularly the diastolic pressure, becomes exceedingly high and the patient complains much of headache, bed rest and one or more phlebotomies of 500 cc may relieve the acute symptoms and tide over the situation for a while Most cerebral vascular accidents result from lesions that cannot be controlled In diabetes, too rigid dieting designed to reduce the blood sugar

<sup>16a</sup> Alvarez, W C "Cerebral Arteriosclerosis with Small Commonly Unrecognized Apoplexies" *Geriatrics*, 1 189, 1916

to normal or the administration of doses of insulin that induce hypoglycemia may initiate a cerebral thrombosis.

With the onset of an acute cerebral vascular accident the

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tion of 4 gr of aminophylline, which may be repeated several times a day clears the sensorium and lessens the paralytic symptoms. Intravenous administration of 25 to 50 cc of 50 per cent glucose solution may serve to reduce intracranial pressure and give some symptomatic relief. If respirations are greatly slowed or Cheyne Stokes in character aminophylline or coramine intravenously and caffeine sodium benzoate in doses of 7½ gr subcutaneously are the best drugs. Beyond this, treatment consists of careful nursing. For the first days the patient will be unable to eat, and fluids should be given intravenously or by hypodermoclysis, particularly if there is high fever. When the patient begins to take fluids or food by mouth, great care should be taken to avoid aspiration of food particles, which results in bronchopneumonia. Bronchopneumonia is common in these patients, from aspiration of saliva and mucus. It should be carefully watched for, and if it appears should be treated as any other case of bronchopneumonia. Owing to the incontinence of urine and stool, bed sores are apt to develop hence the patient should be kept scrupulously clean and dry. At times, particularly in elderly men with prostatic hypertrophy there is retention of urine necessitating catheterization.

In favorable cases improvement is evident within a week. Restoration of function in the paralyzed limbs should always be striven for. The one procedure that hastens such recovery more than any other is attempt by the patient at voluntary use of the affected parts. As soon as possible, therefore, the

patient should be urged to try to move the palsied limbs, and definite exercises should be instituted. Passive movements and massage should be carried out at the same time. With the same end in view, the patient should be moved out of bed at the earliest possible moment. This can usually be done within seven to 14 days after the onset. Far too often these patients are kept immobile in bed for one or more months. Contractions of the paralyzed limbs develop, and subsequent mobilization becomes difficult. Recovery of the leg, in whole or in part, can usually be achieved. Improvement in function of the arm occurs less often. Aphasia, too, may clear up, but if it persists for a week the outlook is not good. Here, too, painstaking re education in speech brings results, and although it will not restore normal speech, it may make it possible for the patient to communicate with the outside world.

During the recovery period, medication is of little value. Sedatives may be given when needed. In certain patients with persistent mental confusion, particularly when there appears to be undernutrition due to a prolonged inadequate diet, the administration of 50 mg of nicotinic acid three times a day seems to help clear the sensorium. This medication should always be tried in older persons with symptoms of senile psychosis or dementia, for in a certain number of cases the mental disturbances are due to a pellagra like dietary insufficiency and not to the cerebral arteriosclerosis.

#### PERIPHERAL ARTERIOSCLEROSIS AND EMBOLISM

Arteriosclerosis of the arteries of the extremities is an important cause of illness and disability in elderly persons. It is the only form of peripheral arterial disease that occurs with any degree of frequency. Thrombo angitis obliterans and acute arteritis are rarely encountered after the age of

50 The age incidence and the relative frequency of its occurrence among the two sexes and among different racial groups are the same as for other forms of arteriosclerosis. Although thickening and tortuosity of the arteries of the upper extremity and of the temporal arteries are common, the lesions rarely lead to arterial closure or symptoms of ischemia. The arteries of the lower extremities are commonly affected with such advanced degrees of arteriosclerosis that obliteration occurs, with consequent impairment of blood supply to the tissues of the extremities. The significant lesion is a collagenous and fibrous intimal thickening causing circumscribed regions of stenosis. Thrombi form in these narrowed arterial sections and extend centrally. The thrombi become organized and canalized and may undergo subsequent hyaline change or atheromatous degeneration. Primary intimal atheroma is not a major feature in the vessels below the level of the popliteal artery. This is in contrast to the usual atherosclerotic lesions of the coronary arteries. Dissection of the arterial tree of legs amputated for gangrene reveals that in this advanced stage there is always widespread occlusion of many arteries. The posterior tibial artery is the one most often occluded, then follow, in the order of frequency, the anterior tibial, the peroneal, the dorsalis pedis and the popliteal arteries. In addition to the intimal and thrombotic lesions, calcification of the media, often with bone formation, is found in almost all cases. This is the so called Monckeberg arteriosclerosis. It does not directly lead to closure of the lumen. It is this medial calcium deposit that makes these arteries visible on the roentgen film.

The pathological and physiological disturbance is quite analogous to that which has been discussed in the section on coronary artery disease. If the development of the arterial disease is slow, collateral channels form that compensate for



the obliterated arteries, so that final closure of the diseased artery may lead to no serious consequences. If arterial closure occurs in the absence of a competent collateral circulation, the tissues that have been deprived of their blood supply die. Clinically we then see gangrene of part of the lower extremity. This is analogous to the myocardial infarction with coronary closure. Closure of an artery of an extremity may be slow owing to gradual progress of the arterial lesion, or it may be sudden owing to local thrombus formation or to the lodgment of an embolus.

Symptoms arising as a result of sclerosis of the arteries of the lower extremity are caused by an inadequate flow of arterial blood. The defective circulation is insufficient to wash away the metabolites that accumulate from muscular contraction, and one substance which Lewis<sup>17</sup> calls the 'P factor' causes pain when it reaches a certain concentration. Just as in coronary disease anginal pain results when the heart muscle is called on for extra work, so in the extremities, the pain of intermittent claudication occurs on effort, when the need for blood of the muscles of the extremities is increased as a result of their use in walking or running. The onset of such pain on effort may be insidious over many years, or it may be sudden if quick thrombosis or embolism of one of the arteries takes place.

Commonly a certain degree of arterial spasm is present in addition to the organic changes in the arterial wall. Relief of this spasm may permit sufficient restoration of the circulation in the extremity to lessen or abolish symptoms due to ischemia. Arterial spasm, however, is encountered less often in arteriosclerotic disease of the arteries of the extremities in older subjects than in other forms of arterial disease that occur at younger ages.

<sup>17</sup>Lewis T. Pickering G. W. and Rothschild P. "Observations upon Muscular Pain in Intermittent Claudication," *Heart*, 15:359, 1931.

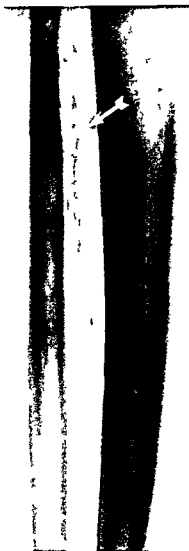


FIG 7 -Calcium deposits (arrow) in the arteries of a leg

*Symptoms* —The presenting symptom of arteriosclerosis of the arteries of the lower extremities is intermittent claudication. On walking the patient experiences a cramping pain, usually in the calf muscles, that compels him to stand still. After a few minutes' rest, the pain disappears, and walking can be resumed until renewed pain again calls a halt. In the early stages this pain comes only on running or on more vigorous exertion, but in the course of time it appears more and more readily, until eventually the patient may not be able to progress more than half a block before being stopped by the pain. Another common complaint is coldness of the legs and feet. Such coldness is due to inadequate arterial blood supply to the leg. Patients with vasospastic disorders have the same complaint, so that this symptom of cold feet should not be regarded as pathognomonic for obliterative arteriosclerosis of the arteries of the extremities. Angina pectoris and intermittent claudication commonly occur in the same patient, and most often it is the pain in the legs, rather than the pain in the heart, that actually stops the patient while walking. Because of the circulatory disorder in his legs, the patient cannot exert himself sufficiently to induce anginal pain. Angina, in him, is provoked by overexerting or by emotional disturbances. Hypertension occurs in about half the cases. As in coronary disease, the hypertension seems to accelerate the arteriosclerotic process, but it is not its direct cause. Diabetes is common in patients with peripheral arteriosclerosis, in fact, nutritional disturbances of the extremities with gangrene are frequent causes of death in diabetics.

When arterial obliteration is far advanced, constant severe burning pain may be experienced in the toes or the foot, it may be excruciating, prevent sleep and yield little to opiates. The parts are very tender, so that even the weight of the bed clothes may be unbearable and walking or standing impos-

sible In an endeavor to find relief the patient may apply a hot water bag or immerse his foot in hot water and so cause a serious ulcer or gangrene Ulcers may appear even without such external injuries Gangrene develops as a result of gradual arterial obliteration or following a thrombosis of a diseased artery The favorite sites are the tips of the toes or the heel A spot of purplish discoloration forms which is very tender Within a few days a black, tough, dry, necrotic eschar forms In the absence of infection it may persist for weeks before it finally sloughs out, leaving a granulating ulcer that takes many more weeks to heal The gangrenous area may gradually spread to involve ever wider regions If it should become infected, a condition of so-called moist gangrene develops which may extend rapidly up the leg Moist gangrene often develops when pyogenic bacteria enter the lesion of a ringworm infection between the toes

*Physical signs*—There may be no abnormality in the appearance of the legs When arterial obliteration is advanced, the leg appears pale, the skin becomes shiny and loses its hair Commonly there are nutritional changes in the toenails, which become thickened, fissured and brittle Trichophyton infection of the skin and nails is common The local resistance of the tissues is low, so that minor cuts and abrasions give rise to slow-healing ulcers and infection readily sets in The foot and leg feel cold, and one can roughly estimate the extent of the arterial obliteration by noting how high up the leg this coldness extends Palpation of the arteries of the leg discloses whether the pulses are open The physician should routinely palpate the dorsalis pedis, posterior tibial and femoral arteries The popliteal artery is difficult to feel When the arteries cannot be felt, or when their pulsations are small, studies with the oscillometer will give a clear picture of the state of the arterial circulation The oscillometer cuff should

be applied on the thigh, on the mid-leg and at the ankle. Normal readings at the mid thigh range from 8 to 15, at the mid-leg from 4 to 10 and at the ankle from 1 to 5. Diminution in the excursion of the oscillometer indicates impairment of the arterial blood supply.

A simple clinical test gives further evidence of a reduced blood supply. With the patient on his back, the leg is elevated to a right angle with the trunk. The patient is then instructed to move his toes and ankle vigorously for a few moments. The leg becomes very pale, almost cadaveric, below the level at which the blood supply is impaired. The patient then sits up and the leg is allowed to hang down. It assumes a dusky bluish-red hue. Roentgen pictures of the legs may reveal calcification of the arteries. This gives no information regarding arterial occlusion, for calcification of the media commonly exists in arteries that are patent. It is frequently noted in the radial and ulnar arteries. However, the presence of medial calcification helps to identify the arterial disease as arteriosclerotic in nature, for calcification does not occur in thrombo-angitis obliterans. Arteriography, the injection of a radio-paque medium directly into the arteries with immediate roentgen visualization, gives a clear picture of the arterial circulation of the leg and reveals obliterated arteries and the extent of the collateral circulation. However, it is a difficult technical procedure, not without danger. It is rarely necessary to carry it out and should be left to experts.

*Treatment*—Great care should be taken to keep the feet and legs clean and free from irritation. Shoes should be well fitting to avoid corns. The patient should be warned against cutting corns and calluses, for infection readily occurs. Tightly fitting garters should not be worn. Epidermophytosis should be carefully treated. Since smoking produces peripheral arterial spasm, tobacco in every form must be abso-

lutely prohibited Even one or two cigarets daily can do great harm Arterial dilators such as the nitrites, theobromine and aminophylline are of no value The intravenous injection of hypertonic salt solution is ineffectual in the treatment of peripheral arteriosclerosis, although it seems to help in cases of thrombo angutis obliterans Intravenous injection of typhoid vaccine, which is often useful in thrombo angutis and in spastic disorders of the arteries, should not be employed in arteriosclerosis The treatment is too drastic and may induce acute thrombosis in a peripheral or a coronary artery Various tissue extracts have been employed Given by mouth, they have doubtful value Given subcutaneously they at times give relief from intermittent claudication The best preparations are Depropanex and Carnacton Injections may be given every other day Mechanical treatment with the pavaex apparatus, that induces alternate suction and compression, is of little value Collens claims good results from his intermittent venous occlusion apparatus Surgical methods of treatment such as ganglionectomy should not be employed in patients with arteriosclerotic vascular disease The operative risk is too great Moreover these procedures relieve only the spastic element in peripheral arterial disease and this is not very marked in arteriosclerotic cases Alcohol injection of the peripheral nerves to relieve pain is dangerous in these patients for it may lead to gangrene Application of mecholyt by iontophoresis gives good results in selected cases

In the presence of dry gangrene the patient should be kept in bed and mummification should be allowed to proceed, in the hope that spontaneous separation of the dead tissues will take place Even if this does not occur, delay gives an opportunity for the development of a collateral circulation and favors better healing of the amputated stump The foot should be placed in a horizontal position, elevation further

be applied on the thigh on the mid leg and at the ankle. Normal readings at the mid thigh range from 8 to 15 at the mid leg from 4 to 10 and at the ankle from 1 to 5. Diminution in the excursion of the oscillometer indicates impairment of the arterial blood supply.

A simple clinical test gives further evidence of a reduced blood supply. With the patient on his back the leg is elevated to a right angle with the trunk. The patient is then instructed to move his toes and ankle vigorously for a few moments. The leg becomes very pale, almost cadaveric below the level at which the blood supply is impaired. The patient then sits up and the leg is allowed to hang down. It assumes a dusky, bluish red hue. Roentgen pictures of the legs may reveal calcification of the arteries. This gives no information regarding arterial occlusion for calcification of the media commonly exists in arteries that are patent. It is frequently noted in the radial and ulnar arteries. However the presence of media calcification helps to identify the arterial disease as arteriosclerotic in nature for calcification does not occur in thrombo angustis obliterans. Arteriography, the injection of a radio opaque medium directly into the arteries with immediate roentgen visualization gives a clear picture of the arterial circulation of the leg and reveals obliterated arteries and the extent of the collateral circulation. However it is a difficult technical procedure not without danger. It is rarely necessary to carry it out and should be left to experts.

*Treatment*—Great care should be taken to keep the feet and legs clean and free from irritation. Shoes should be well fitting to avoid corns. The patient should be warned against cutting corns and calluses for infection readily occurs. Tightly fitting garters should not be worn. Epidermophytosis should be carefully treated. Since smoking produces peripheral arterial spasm tobacco in every form must be abso-

amount of collateral circulation and the degree of accompanying myocardial insufficiency. Embolism occurs chiefly in patients with heart failure, and the inefficient circulation makes the establishment of a collateral circulation more difficult. In favorable cases, the pain subsides, and in the course of 12 to 24 hours color, sensation and movement gradually return to the affected part. There may be complete recovery. If the collateral circulation is inadequate, death and gangrene occur in the tissues that have been deprived of blood. It may involve just the tips of the toes or the whole foot or leg. It is usually a dry mummifying gangrene, unless infection is superadded. Fever and leukocytosis appear. When the embolus is large and is arrested at the bifurcation of the aorta—the so called saddle thrombus—both legs are involved, there is absence of femoral pulsation in both legs, and gangrene may extend up to the mid thigh. If the embolus lodges in the femoral artery, gangrene usually extends above the knee, when the popliteal artery is occluded, death of tissues extends to the mid leg.

Ideal treatment is embolectomy—operative removal of the embolus. This is not often possible. To save the parts distal to the obstruction, operation must be performed within 12 hours of the closure. The patient is often in poor condition because of the basic heart failure and the shock secondary to the embolism, hence the operative mortality is high. Moreover, it is difficult to localize the embolus. In my experience the embolus is always higher than was anticipated. Today, with the use of heparin to prevent clotting, the operative results are a bit better. Since approximately half of all patients with embolism to an artery of the lower extremity recover without the development of gangrene, and since the operative mortality approaches this same figure, conservative treatment is indicated as a rule. The affected extremity is



kept at complete rest and is protected against any and every trauma by a cradle biker. The temperature within the cradle should be thermostatically controlled between 85 and 95 F. For the acute pain, morphine should be given. In addition it is well to give papaverine hydrochloride in  $\frac{1}{2}$  gr. doses intravenously every few hours to release the spastic vasoconstriction. Herrmann has reported good results with the application to the leg of alternate suction and pressure by means of the pavaex apparatus. If dry gangrene develops, conservative treatment should be continued. The part may mummify and eventually drop off. It may take weeks or months for this process to be completed, particularly if some sequestra of bone must be discharged. If a larger area of the leg is involved or if moist gangrene with constitutional reaction develops, amputation becomes necessary. Here it is important to gage the time optimum for operation when the heart failure has been corrected as far as possible, and the patient has recovered from shock. During the interval, absorption from the gangrenous leg may be reduced by placing the leg in a cuff packed with ice that keeps the temperature near the freezing level.

### DISSECTING ANEURYSM OF THE AORTA

This is a rather rare condition more frequent in men than in women. Of the 269 cases collected by Shennan in which ages were known 59 per cent were in persons over the age of 50, although the condition may occur in youth. The underlying cause is a cystic degeneration of the media. Most of the patients have essential hypertension. Usually as a result of sudden physical strain or of excitement there is a rupture of the intima, and blood escapes from the lumen of the aorta into the media. It forces its way between the medial

and adventitial coats and may extend down the whole aorta to its bifurcation or spread up the carotid arteries. The usual termination is external rupture into the pericardium, pleura, mediastinum or retroperitoneal region. In rare cases the force of the blood dissecting its way between media and adventitia splits the intima again and flows back into the lumen of the aorta. If the patient survives, as happens in isolated cases, he is left with a double barreled aorta for the new passage way has become lined with endothelium. Most dissecting aneurysms commence with a transverse irregular tear of the intima in the ascending aorta. But the tear may occur lower down even in the abdominal aorta.

The symptomatology is characterized by intense pain in the anterior and posterior chest which usually radiates down to the abdomen into the legs. In some cases the pain is wholly abdominal. The pain is severe and prolonged and does not yield readily to morphine. Shock develops and this is followed by fever, leukocytosis and dyspnea. If the flow into the iliac arteries is blocked the femoral pulses are absent. Commonly there is anuria due to closure of the renal arteries. This may account for the fact that the blood pressure usually remains elevated. The heart sounds remain of good quality and the electrocardiogram shows no typical changes. Roentgen pictures of the chest may reveal a marked dilatation of the aorta. If the carotid arteries are involved in the process the blood flow to the brain may be stopped and there will be signs of a cerebral vascular insult. The condition usually terminates fatally by complete rupture of the aneurysm in from a few hours to several days. The average duration between rupture and death is four days. Recovery is rare. Treatment is confined to administration of large doses of morphine and absolute bed rest.

## TEMPORAL ARTERITIS

This is a rather unusual, self limited disease that manifests itself with fever, prostration headache and periarteritis of the temporal arteries<sup>18</sup> All cases so far recorded have been in persons over 50 years of age At the onset the symptoms are not specific There are fever, leukocytosis, headache, pains in the muscles and joints After a few weeks the temporal arteries become tortuous and prominent, and tender nodules may appear in the arterial wall Finally the arteries become thrombosed Some of the systemic symptoms are due to involvement of arteries other than the temporals The retinal arteries may be affected, and blindness may result Symptoms simulating brain tumor may be due to multiple cerebral arterial thromboses The disease lasts several months and usually ends in complete recovery Its cause is unknown Pathological examination of the affected arteries reveals inflammation, with many giant cells, of all layers of the arterial wall, thickening of the intima and occlusion of the lumen by thrombosis Lesions have been found in many peripheral and visceral arteries<sup>19</sup> There is no specific treatment Resection of a small portion of the involved temporal artery is said to relieve the headache in many instances, but it does not shorten the course of the disease

<sup>18</sup> Kilbourne E D, and Wolff H G "Cranial Arteritis A Critical Evaluation of the Syndrome of Temporal Arteritis with Report of a Case" *Ann Int Med* 24:1, 1946

<sup>19</sup> Cooke W T et al "Temporal Arteritis A Generalized Vascular Disease," *Quart J Med* 15:47, 1946

## DISEASES OF THE CARDIOVASCULAR SYSTEM— HYPERTENSION

MANY older and even aged persons have blood pressures within the range of what clinicians call normal Table 4, modified from Symonds, gives the average blood pressures of healthy men and women accepted for life insurance The old idea that the systolic blood pressure approximates 100 plus the age of the patient is incorrect Elevated blood pressure always indicates disease, it is not a normal accompaniment of aging

TABLE 4

AGE	MEN		WOMEN	
	S	D	S	D
50-54	131	86	131	87
55-59	134	87	135	88
60 and over	135	87	136	90

The blood pressure is never static, it varies from moment to moment according to the needs of the circulation and the physical and emotional stimuli that are constantly bombarding the vasomotor center During sleep the blood pressure is at its lowest, the lowest level during waking hours is reached when the patient is in bed early in the morning before break-

fast Exercise and digestion are accompanied by rises in pressure Emotional stimuli are effective in raising the blood pressure That is why initial readings taken in a physician's office are often so unexpectedly high A subsequent reading taken with the patient relaxed may be 10 to 50 points lower

### SYSTOLIC HYPERTENSION

By systolic hypertension is meant an elevation of the systolic pressure with little alteration of the diastolic pressure This phenomenon is commonly encountered in old people and is due to a loss of elasticity of the aorta and of the large vessels derived from it The aorta cannot distend to accommodate the volume of blood ejected from the heart, it has no "give" As a consequence, with systole the pressure within the aorta suddenly rises steeply, the blood is rapidly forced to the periphery, so that the pressure rapidly falls, and the diastolic pressure is unchanged The blood pressure may range around 150 to 180 systolic and 80 diastolic Systolic hypertension, encountered when the aorta is dilated and inelastic and accentuated by extensive atherosclerosis, is the result not the cause of a cardiovascular disturbance It does not place an added burden on the heart and does not jeopardize the health or the life of the patient The future of the patient depends on the progress of the underlying atherosclerosis

Systolic hypertension occurs in certain patients with bradycardia The long diastole allows a greater amount of blood to flow from the arteries to the veins, the arteries become comparatively empty, and consequently the diastolic pressure is low while the systolic pressure is unchanged If the cardiac output per beat is increased the systolic pressure is slightly elevated For instance in a patient with intermittent complete heart block, the blood pressure was 130/85 when the

heart rate was 68, when the rate dropped to 20 the blood pressure was 130/40

### ESSENTIAL HYPERTENSION

Essential hypertension is characterized by elevation of both systolic and diastolic blood pressures. The underlying mechanism is a state of peripheral vasoconstriction which retards the flow of blood from arteries to veins and places an increased burden on the heart as well as on the arterial system. The consequences of prolonged essential hypertension, therefore, may be found in the heart, which enlarges and may become insufficient, and in the arteries, which become sclerotic and may give way under strain. The arteries of the heart, kidneys and brain are chiefly affected, and it is in these organs that the complicating lesions in essential hypertension are usually found.

In spite of the brilliant work of Goldblatt and his followers in producing hypertension by clamping the renal arteries, the true cause of essential hypertension in man has not yet been established. Cases in which the hypertension can be directly traced to such an arterial lesion in the kidney and cured by the removal of the kidney are rare. Some other factors must be involved. In certain kidney diseases the hypertension is definitely the result of the renal disorder. The commonest of these diseases is glomerulonephritis which, however, is uncommonly encountered in older people. Hypertension also occurs in periarteritis nodosa with kidney involvement, in congenital polycystic kidneys, in pyelonephritic contracted kidneys and in some cases of urinary obstruction due to prostatic hypertrophy. The kidney lesions in most cases of hypertension are secondary to the hypertension. The long-standing blood pressure elevation causes arteriolosclerosis of

the kidney arterioles with secondary atrophy and fibrous replacement of the glomeruli giving rise to the typical granular kidney, which eventually becomes contracted. In less than 10 per cent of cases of hypertension does this atrophy of the kidney progress to the point where it gives rise to clinical symptoms of kidney insufficiency, with uremia or azotemia.

Hereditary factors play an important rôle. Because hypertension ordinarily becomes manifest at a relatively advanced age, investigation of genetic factors is difficult, but many studies have revealed a high familial incidence of hypertension among the brothers and sisters, parents, uncles, aunts and grandparents of patients with hypertension. In some families every member succumbs to one of the complications of hypertension on reaching the same age period, usually at about 50.

There is no direct relationship between arteriosclerosis and hypertension. At least half of all patients with advanced arteriosclerosis never have hypertension, and many hypertensives do not develop arteriosclerosis of the larger arteries. As a rule, however, hypertensives do develop arteriosclerosis, but the hypertension should be regarded not as the cause of the arteriosclerosis but as a factor that accelerates its development.

Certain cases of hypertension are associated with endocrine disturbances. The most striking are those associated with adrenal cortical tumors. Hypertension is commonly encountered in patients with myxedema. For many years clinicians have sought to relate hypertension in women to the menopause. Hypertension in both men and women develops most often at the age when the menopause takes place, so that the association may be merely accidental. Young women with an artificial menopause due to operative removal of the ovaries or to radiotherapy do not exhibit hypertension. More characteristic of the menopause than a permanent rise in pressure

is a marked lability of blood pressure. This parallels the vasomotor disturbances of the flushes. Hypertension is encountered more frequently in obese persons. It is common in diabetics, occurring in about one third of all those over 50.

There has been growing interest in the so called psychosomatic aspects of hypertension, and some have ventured the claim that hypertension is caused by personality disturbances and can be cured or relieved by psychotherapy. Binger and his associates published a careful study of this matter.<sup>1</sup> They found that hypertensives have neurotic personalities and have suffered from insecurity in their childhood and an unsatisfied dependent relationship to a threatening parent. Binger believes that hypertensives differ from other persons with neurotic disorders in as much as they finally submit to the hostility of the parent figure and accept defeat of their own aggressive drives. This leads to tenseness, anxiety states and depressions. They develop compensatory aggressive drives but remain insecure. Anything that threatens their security increases their anxiety and depression. Much of this anxiety is absorbed in their symptoms; they rarely develop organized neurotic symptoms. There is no proof that this type of personality disturbance is the cause of hypertension; they are associated phenomena that have a common cause. Moreover, not every hypertensive need exhibit these character traits, and not everyone who has such a personality disturbance has elevated blood pressure.

*Pathology*—The characteristic lesion associated with hypertension is an arteriolosclerosis found chiefly in the arterioles of the kidneys, spleen and pancreas. Arteriosclerosis of the larger arteries is not constant. Cardiac hypertrophy is found in almost every case in which the diastolic pressure is

<sup>1</sup> Binger, C. A. L., et al. *Personality in Arterial Hypertension* (New York City: American Association for Research in Psychosomatic Problems, 1945).





FIG. 8—Extreme hypertrophy of the left ventricle in a patient with hypertension and leniplegia

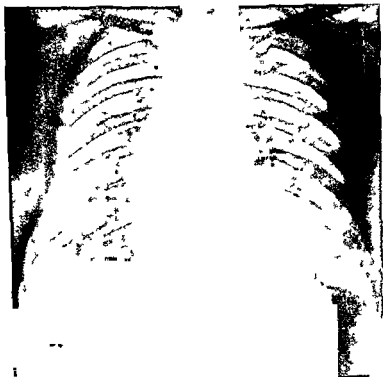


FIG 9—Enlargement of the left ventricle and generalized dilatation of the aorta due to essential hypertension

persistent above 100. The left ventricle is first involved but right ventricular hypertrophy follows sooner or later. These hearts may attain a tremendous size. Coronary artery disease may or may not be present. Myocardial lesions depend on the state of the coronary arteries. The kidneys may be normal or they may show various degrees of arteriosclerosis with contraction. Malignant sclerosis is uncommon in older persons. Cerebral hemorrhage, a common complication of hypertension, is determined in many instances by the presence of military aneurysms of the cerebral arterioles. These occur most frequently in the vessels of the corpus striatum and it is here that hemorrhage most often occurs.

### SYMPTOMS

Significant hypertension is rare under the age of 40, commonest between the ages of 45 and 70 and less frequent again among the higher age groups. For persons with hypertension usually die before they reach old age. The condition is distinctly more common among women than among men.

Often hypertension is discovered accidentally in the course of a routine physical examination of patients who are unaware of any deviation from normal health. The elevation in pressure in such cases is usually not great, maybe 175/95. There is no cardiac enlargement and no evidence of renal disturbance. Measurement of the blood pressure of such a patient at different times of the day under varying conditions over a long period of years will demonstrate that it is undergoing constant and great fluctuations. Early in the morning the pressure is apt to be low; in the afternoon it is high. Emotional upsets and mental strains are quickly reflected in a rise in pressure. It is the systolic pressure in particular that is labile. In these patients apparent cures are seen with par-

ticular frequency, under treatment or following an acute intercurrent infection, the pressure at times returns to normal. But the drop is only temporary. In the course of weeks and months the pressure again rises and the former clinical picture is repeated. In the early stages of the disease, therefore, the variability rather than the absolute height of the sphygmomanometric readings is of the greatest significance.

This state of affairs may continue for five, 10 or even 20 years without much change and few symptoms. More often, however, there is a slow but steady tendency of the pressure to rise to higher levels. With this it gradually becomes more stable, the fluctuations are no longer so extreme, and normal figures are rarely approached. It is at this stage that the diastolic readings reach 105 and over and systolic readings 200 or more. Now the patient has arrived at the stage of fully developed hypertension. Hypertrophy and dilatation of the heart, with possibly relative mitral insufficiency, have become manifest, and certain subjective complaints have appeared.

Many patients first seek medical attention at this stage. Among the earliest symptoms are headache, vertigo and shortness of breath on exertion. Nocturia is not infrequent, although the kidney function is rarely impaired. This nocturia is of cardiac origin from passive congestion of the kidney. During the night's rest the circulation, and with it the renal secretion, improves. The nocturia of Bright's disease is due to inability of the kidney to concentrate the urine, so that large quantities of water must constantly be excreted to wash out the solids. The two conditions may be differentiated by the fact that in nocturia of cardiac origin the specific gravity of the urine is high, and in that of renal origin it is low.

which, in most instances, can be readily identified. Epistaxis

in a person over 40 years of age should always suggest the possibility of an existing hypertension

*Eyegrounds*—All patients with hypertension show changes in the eyegrounds. The earliest and most constant sign is narrowing and increased tortuosity of the arteries. The degree of narrowing roughly parallels the level of hypertension. In more advanced cases, particularly when there is rapid progress of the disease—the so called malignant hypertension—there are hemorrhages and white scars in the retina. Papillitis is seen only in malignant sclerosis and this is uncommon in older persons.

For convenience of description the further symptomatology of essential hypertension is best discussed under several captions determined by the presenting symptoms. The three chief forms of the disease may be termed the cardiac, the cerebral and the renal.

*Cardiac form*—Symptoms referable to the heart dominate the clinical picture in over 50 per cent of cases. The earliest sign is dyspnea on exertion. This may at first be of slight degree. The patient is apt to be plethoric, his face red or slightly cyanotic, the superficial veins in the neck and on the dorsum of the hand swollen. Examination reveals a heart that is enlarged, especially to the left, with the sounds of good quality and the aortic second sound accentuated. A systolic murmur due to dilatation of the aorta is commonly heard at the aortic area. Not infrequently, an increased area of aortic dullness can be mapped out by percussion and demonstrated by the roentgen ray. The pulsation of the aorta or innominate artery may be felt in the suprasternal notch, and if the subclavian arteries share in the dilatation their pulsation may be visible above the clavicles. The accentuation of the aortic second sound in these cases is due in part to the fact that the dilated aorta is longer than normal and being fixed at its root, be-

comes more closely approximated to the anterior chest wall

The electrocardiogram shows left ventricular preponderance when the left ventricle has become enlarged. In later stages of left ventricular hypertrophy the T waves in lead I, or in leads I, 2 and 4, may become negative. There may be a concomitant depression of the ST segments in these leads, and the QRS complex may be widened to 0.11 or 0.12 second. Such findings may be due solely to left ventricular strain and need not connote coronary artery disease.

Sooner or later *cardiac insufficiency* becomes more marked and the systolic apical murmur of a relative mitral insufficiency becomes manifest. Evidences of left ventricular failure dominate the clinical picture. Dyspnea is the most striking symptom and is caused by engorgement of the pulmonary blood vessels. At first, shortness of breath appears only on exertion, but soon it persists when the patient is at rest. Congestive râles may appear in the lower lobes of the lungs. In many cases nocturnal paroxysmal dyspnea becomes a distressing symptom, which in its severest form is accompanied by pulmonary edema. It does not differ from the similar condition observed in patients with coronary artery disease or with syphilitic aortitis, for acute left ventricular failure is the cause of such paroxysmal dyspnea in all forms of heart disease. *Gallop rhythm*, heard at the apex, is characteristic of the failing heart of hypertensives. In the earlier stages of cardiac insufficiency it may appear only after exercise and so may become a valuable indicator of impending cardiac failure. With gallop rhythm the heart rate is rapid and the pulmonic second sound is accentuated. When the left ventricle fails the right ventricle too labors at a disadvantage, and soon signs of right heart failure appear. The neck veins become engorged, the liver enlarges and ascites or edema of the legs may develop.

In the vast majority of patients with hypertension, the onset of severe myocardial insufficiency is accompanied by a fall of some 20 to 40 mm in the systolic pressure, with no alteration or with a rise in the diastolic pressure. When the myocardium weakens, the systolic output of the ventricles diminishes, and therefore the systolic pressure will be lowered, but this does not affect the tonus of the peripheral arterioles which determines the diastolic pressure. A small pulse pressure results, which in hyperpiesis is pathognomonic for heart failure. An arterial blood pressure reading of 160/120 or of 130/100 clearly signalizes the presence of serious myocardial weakness.

The popular conception that the blood pressure in patients with hypertension drops to normal or subnormal figures when the heart fails is false. This occurs only as a terminal phenomenon a few hours before death or with widespread infarction of the heart from closure of a main stem of the coronary arteries. A marked drop in diastolic pressure is due not to cardiac failure but to a diminution or loss of the factor which determined the increased vascular tone.

Alterations in cardiac rhythm are not uncommon. The irregularity most frequently encountered is that due to extrasystoles. They are rarely of much significance and do not affect the ultimate outcome of the case unless they are numerous and persistent. Auricular fibrillation occurs in about 7 per cent of hospitalized cases, but in far fewer ambulant patients. The onset of auricular fibrillation is followed by the usual evidences of heart failure, which can be controlled by the proper administration of digitalis.

Alternation of the pulse is common when the heart fails, but it is rarely recognized because it is rarely sought for. In few cases is it sufficiently marked to be capable of detection by the unaided finger. In many it can be discovered by means

of the sphygmomanometer When the first arterial sound becomes audible, as the air in the cuff is gradually released, the rate of the sounds will be exactly one half the heart rate, for only every second beat is strong enough to come through at the systolic pressure As the pressure in the cuff is lowered 10 to 20 mm more, all the pulse beats come through suddenly, and the rate of the sounds is doubled Alternation is, in most instances, of grave significance The slower the pulse rate associated with alternation, the graver the prognosis Coronary artery sclerosis in all of its manifestations is commonly associated with hypertension, but the relationship between these two conditions is not causal or constant, except that the hypertonia may contribute to the coronary artery disease In a certain number of cases an attack of substernal oppression may be the first symptom leading to the discovery of latent hypertonia Many hypertensives die of cardiac infarction

*Cerebral symptoms*—In a large number of cases the symptoms of hypertension are manifested by cerebral vascular disturbances The earliest symptoms are those of a transient disturbance of cerebral function Headaches, sudden vertigo and confusional states which pass off rapidly are not uncommon Again there may be sudden aphasia or merely an impediment of speech which quickly disappears Other patients experience transient true hemiplegias A woman, 52 years of age had known for seven years that her blood pressure ranged about 180 One day she awoke, vomited and found herself unable to talk or to move the right arm or leg Within about an hour speech as well as slight movements of the paralyzed limbs began to return, and within about 12 hours all of the symptoms had disappeared with the exception of a severe headache Twenty years later, although the moderate hypertension persisted, there was no evidence of



any cerebral lesion, nor had there been a repetition of the attack. She finally died of a massive cerebral hemorrhage at the age of 77. Not infrequently an attack of hemiplegia is the first indication of the existence of hypertension. Many of my hospital patients claim that they were well before their stroke. The cerebral manifestations may take any of the forms described in the section on arteriosclerosis of the cerebral arteries.

*Renal symptoms*—Symptoms due to disturbed kidney function are infrequent. Only about 6 per cent of patients with essential hypertension succumb to uremia. The presence of traces of albumin and casts in the urine is of little clinical significance. The most important early sign of kidney insufficiency is inability of the patient to pass concentrated urine. A simple concentration test will unmask this condition. The patient is given no fluids after 6:00 P.M. The following morning he discards the first specimen of urine passed. Specimens are then collected at 9:00, 10:00 and 11:00 A.M., the patient in the meantime receiving no food or drink. A normal kidney under such circumstances will secrete urine of a specific gravity of from 1.025 to 1.030. The degree of damage to the kidney is indicated by the specific gravity. If the specific gravity does not exceed 1.015, there is considerable impairment of function, but azotemia does not develop until the specific gravity becomes fixed at between 1.012 and 1.010. Azotemia is recognized by finding an increase in the urea nitrogen of the blood. The normal figure is 15 mg. per 100 cc. of blood. With kidney insufficiency, the level of urea nitrogen rises gradually and may reach as high as 150. Severe cardiac insufficiency, by causing passive congestion of the kidneys and thus interfering with their function, may be accompanied by moderate retention of urea nitrogen. Figures up to 40 or 50 mg. per cent may be encountered. This will clear up if cardiac compensation is restored.

## PROGNOSIS

Patients do not die of hypertension as such but as the result of the damage to various organs particularly the heart arteries and kidneys caused by the prolonged excessive strain to which the cardiovascular apparatus is exposed. Many years may elapse before such injury disturbs the normal mechanism of the body and the resistance of the several organs of different individuals to the insidious effect of the hypertensive state varies tremendously. It is difficult therefore to generalize about the prognosis of hypertension.

The younger the patient the worse the outlook. Persons under 45 with marked hypertension rarely survive more than a few years. Older individuals may survive five, 10 or even 20 years in spite of persistently high blood pressure and symptoms pointing to beginning cardiac weakness or disturbance of the cerebral circulation. Hypertension in persons over 70 is well borne and often gives rise to no complaints or symptoms. The prognosis is much less favorable in men than in women.

The diastolic blood pressure is of great significance in estimating the seriousness of the case. A diastolic pressure which is persistently above 130 mm Hg is incompatible with survival for more than a few years and pressures that reach 150 predicate an early end. Blood pressure readings taken from time to time in the physician's office may be very deceptive. Emotionally tense patients may exhibit such high readings at every visit that the physician wonders how they survive. I have patients who show pressures of 300/150 for years. If one measures their blood pressure in their homes when they are relaxed one finds that the actual levels for most of the day are much lower.

In patients with hypertension the causes of death in their

order of frequency are cardiac failure, cerebral vascular insults, coronary artery disease and uremia

To a certain extent the early symptoms complained of by the patient foretell the manner of his death. Thus the majority of those in whom symptoms of myocardial weakness occur early die eventually from cardiac failure. Those with angina pectoris are apt to die of cardiac infarction. Headache suggests the probability of death from apoplexy or more rarely uremia.

Clearly defined evidence of disturbed renal function such as fixation of the specific gravity of the urine and retention of urea in the blood, provided it is not conditioned by cardiac failure, is always of grave significance. Death may be expected within a year.

The majority of patients with hypertension, however, survive many years, particularly those whose disorder is discovered before the appearance of symptoms. Their recuperative power after the onset of severe myocardial insufficiency is often so little short of marvelous that no patient should be given up as hopeless. After an attack of apoplexy they may survive many years. A good deal depends upon the mode of life of the individual. If he can spare himself and remain under careful and prolonged medical supervision, many years can be added to his existence, even though he continues at his usual occupation.

The earlier stages of hypertension are characterized by a purely functional disturbance, an increased tonus of the arterioles which although anatomically normal, are set, so to speak, in the position of partial contraction. With this there is marked instability of the vegetative nervous system which leads to great and frequent changes in arteriolar tone with parallel variation in arterial blood pressure. The early phases of arterial hypertension are marked particularly by instability

of the blood pressure and by disturbances in the domain of the involuntary nervous system. It is only after many years that the secondary changes in the heart and arteries and in the kidneys occur.

### MANAGEMENT

The spontaneous wide variations in blood pressure have led to many false therapeutic claims. Worry, anxiety or excitement may lead to marked transient rises in blood pressure, rest and relaxation will induce a drop. If the patient is allowed to rest on a comfortable examining couch and blood pressure readings are made at short intervals, the later readings will always be lower than the initial ones. Not infrequently the systolic pressure may drop as much as 20 to 40 millimeters of mercury. Just as the estimation of the metabolic rate is made with the patient under basal conditions, so in taking comparative blood pressure readings the attempt should be made to obtain readings under circumstances that are as uniform as possible and that eliminate factors which may induce transient elevations in pressure. Although in the daily practice of medicine it is rarely possible to obtain blood pressure readings with the patient in the fasting, resting stage, observations can be made with the patient relaxed, and repeated readings can be made for five or 10 minutes until the blood pressure becomes more or less stabilized at a level which is ordinarily considerably lower than the initial one.

It is this great variability of blood pressure and its tendency to fall spontaneously that makes difficult the evaluation of different forms of therapy and that has led to so many fads in the treatment of arterial hypertension. Innumerable drugs have been exploited with enthusiasm for a few months or years, only to be discarded and forgotten.

*Prevention*—Arterial hypertension cannot be prevented so long as we are in complete ignorance of its cause. It has repeatedly been claimed that the hurry and stress of our mode of living leads to arterial disease and hypertension and that it is responsible for the increasing mortality from these conditions. Careful analyses have proved the incorrectness of this view, so that the hope that arterial hypertension might be prevented by a return to the simple life is without foundation.

Improper diet, excessive use of alcohol and tobacco, absorption of toxins from a sluggish colon and focal infections have severally been considered causative agents of arterial hypertension. There is no evidence in support of these views. Although high blood pressure is commonly encountered in obese individuals, there is no direct connection between obesity and hypertension. It seems more probable that some underlying constitutional disorder lies at the root of both conditions.

It is becoming more and more apparent that hereditary factors are so important that arterial hypertension should be regarded as a familial disorder. The discovery of a case of hypertension should call for periodic check-up of other members of the family—brothers and sisters, parents and children. This will not prevent the development of the disorder, but early recognition and appropriate treatment may help to minimize its ill effects and avoid or postpone the more serious complications. Similarly, periodic health examinations will reveal many instances of arterial hypertension in the asymptomatic stage and enable the subject to care for himself properly.

*Treatment*—An individual who has no symptoms, in whom elevation of blood pressure is accidentally discovered, requires no active treatment. He should be told that his blood pressure is a bit high, but the physician should explain that as

a rule hypertension is well tolerated for years and that it calls for the avoidance of excesses of all kinds and the establishment of sensible habits of living. The exact level of blood pressure should not be revealed to the patient and the natural spontaneous variations in pressure should be made clear. Many patients become slaves to the sphygmomanometer and are elated when the reading is 10 mm lower and become depressed and apprehensive when it is a few points higher than at a previous examination. The blood pressure should not be measured too frequently.

The earliest symptoms of hypertension are headache, vertigo, flushes, sticking precordial pain and palpitation. These symptoms are related to the disturbance of the vegetative nervous system and do not, as a rule, reflect organic disease.

In the early stages of the disorder treatment should concern itself with regulation of the patient's life and with allaying his fears. The patient should discipline himself to moderation in every bodily and mental activity: temperance in eating, drinking, smoking, exercise, work and pleasure. If he can learn to slow the tempo of his living and go through his daily routine without undue fuss or hurry, his capacity for work and accomplishment will remain large and his activities will actually be beneficial to him by maintaining his morale.

With these precautions it is best for the patient to continue at his work. In the case of the business executive, the professional man or the clerical worker, the occupation involves little physical strain. Too great responsibilities leading to undue anxiety and worry should be avoided as far as possible. The patient should learn to temper the drive of his ambition even at the cost of lessening his accomplishment. Most factory hands, employed at machines, can carry on. It is the heavy type of labor of the unskilled workman that is harmful.

Whenever possible the patient should plan for at least two

vacations during the year. The vacation should provide physical rest, a change of scene and new interest. Moderate exercise is beneficial. Walking, even long cross country hikes, offers the best form of physical activity. Golf and horseback riding are permissible. More violent sports such as tennis, handball and baseball, in fact all competitive games in which the participant drives himself to win, are too strenuous. Bathing and leisurely swimming may be allowed but should not be overdone. The best check on the amount of exercise that can be borne without harm is the reaction of the patient himself. Exercise should be followed by a sense of well-being and should never be carried beyond the threshold of dyspnea and fatigue.

**Diet.** The diet should be liberal. No particular food restriction will reduce the blood pressure or favorably influence the course of the disease. It is still necessary to emphasize that protein is not harmful to persons with hypertension, even

persons. Many patients with hypertension, either on their own initiative or on the advice of their physician, avoid all protein intake for months and years and in time suffer from protein starvation. This may be so extreme as to lower the blood proteins and favor the occurrence of edema. It is not uncommon to encounter moderate edema of the legs, great weakness and some dyspnea in patients with arterial hypertension, in whom careful investigation reveals no heart failure and no nephritis. Such patients may be incorrectly treated for months with cardiac stimulants and diuretics and kept in bed, but without improvement. Inquiry will usually reveal protein starvation. The administration of a high protein diet, 100 to 125 Gm. a day, and large doses of iron, for there is usu-

ally secondary anemia, will lead to complete relief of symptoms within a few months. Salt restriction, which has been advocated by some, is of no value in the absence of edema.

Coffee and tea in moderation are not harmful if they do not provoke sleeplessness. Strong alcoholic drinks and drinking to excess are inadvisable because of the vasomotor reactions which they induce. A glass of wine or sherry often acts beneficially in promoting relaxation and allaying restlessness and irritability. Smoking is permissible.

In the presence of obesity the diet should be adjusted to in

the fat and carbohydrate intake (see p. 42). Eating is largely a matter of habit, and if the patient is made to understand the importance of such an adjustment of diet he will soon learn to be content on the restricted regimen. The first few weeks are difficult and are often marked by real hunger and weakness. At times a few pieces of candy between meals or the administration of 2.5 to 5 mg. of benzedrine sulfate tablets before meals tides the patient over the first weeks until he has become accustomed to the diet.

The bowels should be regulated. The administration of 3 gr. of calomel at night followed by  $\frac{1}{2}$  oz. of magnesium sulfate in the morning may relieve headache and vertigo. This medication should not be repeated oftener than once every two to three weeks.

**Drugs.** No drugs will permanently lower the blood pressure. The nitrites, even those with longer lasting effects, such as erythrol tetranitrate, induce only transient lowering of pressure, and their by effects usually make the patient more uncomfortable. Arterial dilators such as theobromine and metaphylline are also of little benefit, as are the various muscle and pancreatic extracts. Potassium thiocyanate tends



to reduce blood pressure and relieve the headaches and nervousness of hypertensive patients. It is given in doses of from 5 to 15 gr. a day, so adjusted as to maintain a blood level not exceeding 10 mg. per cent. Some authors report favorable results, but toxic reactions such as skin eruptions, weakness, nausea and vomiting are common, and over one half of the patients, particularly those in the more advanced stages of the disease, experience no benefit. I have found little occasion to employ this drug.

Maintenance of lower levels of pressure is best achieved by controlling the exaggerated nervous and emotional reactions of the patient. Here sedatives are very useful. The best of all is the classic mixture of chloral hydrate and sodium bromide. The dosage should be adjusted to the individual from 5 to 10 gr. of chloral hydrate and from 10 to 20 gr. of sodium bromide, in a vehicle such as aromatic elixir given two or three times a day. The barbiturates, too, are very useful. The usual dose is  $\frac{1}{2}$  gr. of phenobarbital three times a day.

In the early stages of essential hypertension it is not necessary or advisable to give treatment directed to the heart. Some have advocated giving tonic doses of digitalis to strengthen cardiac contraction and to prevent heart failure, but this is of questionable benefit. Potassium iodide is of no value and does not prevent the development of arterial disease. The administration of estrogenic hormone to hypertensive women with vasomotor disturbances of the menopause may give symptomatic relief and control marked fluctuations of the blood pressure. It does not permanently restore the blood pressure to normal. Diethylstilbestrol may be given in a dosage of 0.5 mg. daily, or else estradiol benzoate in oil, 2,000 rat units by injection, three times a week. None of the other hormones are helpful.

Any measure that promotes muscular and mental relaxa-

tion tends to lower the blood pressure. Warm baths at a temperature of about 100 F. are often very soothing. These are best taken before retiring at night or before an afternoon siesta. Electrotherapy may have a similar effect. Some have had success with exercises planned to induce muscular relaxation.<sup>2</sup> Occasional one or two week periods of bed rest are probably more valuable than all of the other measures combined. But care should be taken so to plan these enforced vacations that the benefit from the body rest is not counteracted by worry and irritation at the inactivity.

**Surgery** In recent years a number of operations to lower blood pressure have been devised. The rare cases of paroxysmal hypertension due to suprarenal tumors can be cured by operative removal of the tumor.<sup>3</sup> Peet<sup>4</sup> has devised a technic of bilateral supradiaphragmatic splanchnicectomy with lower dorsal ganglionectomy. He claims that one of every three patients with malignant hypertension who are operated on survives five years or longer and that many others experience reduction in blood pressure and symptomatic improvement. White and Smithwick<sup>5</sup> report less favorable results with this procedure and Smithwick now uses a combined supra and infradiaphragmatic approach with removal of the sympathetic trunk from the tenth dorsal to the third lumbar with excision of the great splanchnic nerves. The operative mortality is small. This operation produces marked postural hypotension which gradually disappears in the course of a

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<sup>4</sup>Peet M M Woods W W and Braden S "The Surgical Treatment of Hypertension" J A M A 115 1875 Nov 30 1940

<sup>5</sup>White J C and Smithwick R *The Autonomic Nervous System* (2d ed New York City The Macmillan Company 1941)

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few months Smithwick obtains favorable results in most cases in which the x-rays do not show advanced changes and in which preoperative testing with large doses of sodium amytal induces a fall of blood pressure to normal. Reduced renal or cardiac function and cerebral vascular damage in general are contraindications to operation for in such cases the results are poor. Most patients over the age of 50 do not respond favorably and are poor operative risks. Although undoubtedly brilliant results have been achieved in individual cases the ultimate value of these operations is still an open question. Many years will elapse before one can judge whether they prolong the lives of hypertensives for the course of hypertension is a long one. The patients who respond best to operation in general are those with the best outlook under a medical regimen. For the present I should be inclined to restrict operation to young individuals with progressive severe hypertension but with good kidney function.

*Complications*—There are a number of symptoms that are particularly troublesome to both the patient and the physician. Chief among these is headache which should be treated by the general measures that have been described. Caffeine or aminophylline often relieves hypertensive headache. If these are ineffectual and particularly if the blood pressure is rising or is at a very high level and if bed rest brings no relief a phlebotomy of about 500 cc. should be done. This often abolishes the headache immediately and remarkably enough the respite may last for several months. Blood letting in such cases not alone ameliorates symptoms but may prevent the occurrence of a cerebrovascular accident. Severe persistent headache with a mounting diastolic pressure always calls for active treatment for it is often a warning of impending apoplexy.

Tinnitus aurium may be very disturbing. One can only

employ the general measures that have been outlined, but often they are ineffectual. Vertigo may at times be relieved by wearing a well fitting corset or abdominal binder that pulls in the lower abdomen.

Kidney insufficiency is a relatively unusual complication of essential arterial hypertension. When true uremia sets in treatment is of little avail. More often oliguria and slight azotemia result from heart failure and may recede as a result of supportive measures directed to the heart.

Heart failure may be due to gradual flagging of an over-taxed myocardium, or to complicating disease of the coronary arteries. Its treatment does not differ from that of heart failure due to other causes.

## **DISEASES OF THE CARDIOVASCULAR SYSTEM— SYPHILIS**

SYPHILIS is the cause of about 10 per cent of heart disease in persons past the age of 50. Syphilis of the myocardium is rare, the common lesion is in the aorta. Routine examination discloses that about 10 per cent of individuals with late syphilis have syphilitic involvement of the cardiovascular system. More intensive study with roentgen rays reveals an incidence of some 33 per cent, and autopsy studies indicate a still greater frequency of aortic involvement in males with late syphilis. These data point to the necessity of searching for cardiovascular syphilis in every known syphilitic and, in view of the late appearance of signs of cardiovascular syphilis—on the average 20 years after the initial infection—of repeating examination annually. Cardiovascular syphilis is often associated with cerebrospinal syphilis and tabes and paresis.

The frequency of syphilis in any group of individuals depends on the classes of society from which they are recruited. In the male wards of a city hospital, positive Wassermann reactions may run as high as 25 per cent. On the other hand, among applicants for license to peddle in New York City, who were subjected to routine Wassermann tests, 8.4 per cent were found positive.

Syphilitic disease of the aorta is four to five times as common in men as in women, and twice as common in Negro as in white patients

The symptoms of syphilitic aortitis appear in the majority of patients between the ages of 45 and 50. Acquired syphilitic aortic disease may occur as early as age 25 or as late as age 70. The period elapsing between the contraction of the chancre and the development of symptoms of aortitis is, on the average, 20 years. At what time actual infection of the aorta occurs is unknown, but it must antedate by many years the onset of typical symptoms. The shortest period between the primary infection and death from syphilis of the aorta is one year; the longest 55 years. I have a patient who had a chancre at age 20 and who first developed syphilitic aortitis at age 58.

Syphilitic aortitis is localized most commonly in the ascending aorta, particularly in the suprasigmoid portion, but it often involves the whole arch and not infrequently the entire thoracic aorta. Syphilitic lesions of the abdominal aorta are rare. As a rule the process stops sharply at the descending aorta or at the diaphragm.

#### PATHOLOGY

The inflammation begins as a round cell infiltration around the vasa vasorum in the adventitia and media. Gradually these areas enlarge and extend farther into the media. With this, the elastic and muscular layers of the aorta are destroyed and mononuclear, epithelioid and giant cells surround the necrotic areas. These, in fact, constitute minute gummas. Gradually this syphilitic granulation tissue invades larger and larger areas of the media and extends through to the intima. In the course of time scarring takes place, and with this the wall of the aorta becomes thinned and the



intima is drawn in and puckered over the scarred areas. This pathological process produces a characteristic gross appearance of the involved aorta. The aorta is usually dilated and the intima is wrinkled, often with longitudinal striations.

Atherosclerosis of the aorta is distinguished from syphilis by the fact that in the former the intima is primarily affected. There is lipoid infiltration of the intima with secondary calcification, ulceration and mural thrombosis. The lesions are not confined to the thoracic aorta, but extend down beyond the bifurcation into the iliac arteries. Frequently the two lesions, atherosclerosis and syphilis, are combined and it may then be difficult to recognize the syphilitic lesions with the naked eye.

In many instances the pathological process surrounds the mouths of the coronary arteries in the sinuses of Valsalva and may proceed to complete occlusion of the orifices. Interference with the coronary blood supply results in infarction and scarring of the myocardium. Syphilitic involvement of the coronary arteries themselves is very rare.

The most common extension of the syphilitic process is to the aortic valves. The lesion is quite characteristic. The distortion of the wall of the aorta at the insertion of the valves leads to a widening of the commissures between the valve leaflets. As a result the free margins droop and become everted and in the course of time they become thickened or rolled. This leads to insufficiency of the aortic valves.

Aneurysms of the aorta, with few exceptions, develop on the basis of a syphilitic aortitis. From 80 to 90 per cent of aneurysms of the thoracic aorta are syphilitic in origin. Their location corresponds to the site of predilection of syphilitic infection of the aorta. Boyd,<sup>1</sup> in an analysis of 4 000 aneurysms

<sup>1</sup> Boyd, L. J. "Study of 4 000 Reported Cases of Aneurysm of the Thoracic Aorta." *Am J M. Sc.* 163: 654, November, 1924.

found the ascending aorta, the arch, the descending and thoracic aorta involved in the ratio of 10 7 3 1. In very old individuals extensive atherosclerosis may cause aneurysms. These are often multiple and most commonly arise from the abdominal aorta, although rarely they may be thoracic. The average age at death in 20 cases collected by Ruffin was 72.7 years. One third of the patients died of rupture of the aneurysm. Other rare causes of aneurysms are rheumatic fever, tuberculosis of the aorta and trauma. We shall confine our description to syphilitic aneurysms.

An aneurysm is a localized dilatation of the arterial wall. It may be diffuse and fusiform, from 6 to 10 cm. long, or more commonly it may be saccular. Aneurysms arise as a result of the injury to the elastic and muscular tissue of the media by the syphilitic process. The wall of the aorta gives way in the weakened areas, but the wall of the aneurysmal sac is not simply the dilated aortic wall. If one traces the several layers of the aortic wall from the healthy aorta into the sac, one finds that the intima and media stop abruptly at the entrance to the aneurysm. The wall of the sac is made up of newly formed white and elastic connective tissue into which have been fused not alone the adventitia but any other structures with which the aneurysm comes in contact during its growth. Thus veins, the mediastinal fascia and even bronchi may be incorporated into the wall of the sac.

The inner surface of the sac tends to be covered with endothelium, but often is thickened and calcified. Thrombi commonly form within the sac, and these as a rule become laminated and organized, thus serving to strengthen the wall.

As the aneurysm grows it impinges on the mediastinal structures that surround it and causes secondary changes in them. Thus the vertebrae, ribs or sternum may be eroded, the bronchi may be compressed, with secondary bronchiec-

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FIG 11—Arteriosclerosis of the aorta. Note crescentic shadow at the aortic knob representing a cross section of a calcified plaque.



Fig 12.-Syllil tue rt lis lclt roentgenogram fusiform dilatation of the aorta right pathologic specimen of the same case

tasis, the recurrent laryngeal nerve is often destroyed and the esophagus may be displaced and partially occluded

Rupture of the aneurysmal sac causes death in about one half of the cases. This occurs most frequently into the pericardium, pleura or lung, pulmonary artery or superior vena cava. The aneurysm may rupture externally.

The heart itself, as has been pointed out, rarely shows any

huge aneurysm. When the coronary arteries are occluded, the usual phenomena of cardiac infarction occur. With aortic insufficiency, dilatation and hypertrophy of the left ventricle take place and are often marked. In the later stages the other chambers of the heart also undergo enlargement.

### SYMPTOMATOLOGY

The clinical manifestations of syphilitic aortitis are best discussed in several categories determined by the presence of certain complications: simple uncomplicated aortitis, aortitis with aortic insufficiency, aortitis with involvement of the mouths of the coronary arteries and aortitis with aneurysm of the aorta.

*Simple aortitis*—In the absence of one of the complications just enumerated, syphilitic inflammation of the aorta gives rise to no symptoms. Fully one quarter of cases discovered at autopsy are unsuspected during the patient's life. Much has been written concerning aortic pain, so called aortalgia, and attempts have been made to distinguish this particular type of pain from other forms of cardiac pain. It has been described as a continuous pressing pain behind the manubrium. There is no valid evidence for these views, for pain does not



FIG 13 -Syphilitic aortitis sacular aneurysm of the aorta

occur in simple syphilitic aortitis. Paroxysmal dyspnea, too, does not torment the patient with uncomplicated aortitis, but is experienced only if there is an accompanying insufficiency of the aortic valves, hypertension or aneurysm.

The diagnosis must be based on the history, the physical signs and the roentgen examination. Because of this, most cases are missed until some complication has supervened. The discovery of simple syphilitic aortitis is usually accidental or else the result of a careful examination of a known syphilitic. This is very unfortunate, for the only hope of successful therapy lies in early diagnosis and treatment. It is, therefore, exceedingly important to subject persons infected with syphilis to periodic examinations in order to discover any lesion in its earliest stages.

The physical signs are those of aortic dilatation and elongation and do not differ from those characterizing atherosclerotic or other forms of aortic disease. Even when the physical signs are definite the diagnosis in the last analysis depends on the known presence of syphilis, as revealed by the Wassermann reaction or other data as well as on the age of the patient.

Syphilitic aortitis involves chiefly the ascending portion of the arch and results in dilatation and elongation of the vessel. It is the elongation more than the dilatation that gives rise to physical signs. The aorta approaches closer to the anterior chest wall and extends to the right of the sternum. It also ascends in the thorax, so that its pulsation may be felt by inserting the finger in the suprasternal notch. In its ascent it carries with it the origin of the innominate artery, so that the right subclavian rises and its pulsation can often be felt above the clavicle. It is only in rather extreme dilatation that the aorta extends to the left of the sternum.

On palpation one may note pulsation in the second inter-



space to the right of the sternum as well as the diastolic shock accompanying the closure of the aortic valves. As Hoover pointed out the muscular sense of the muscles which lead from the head to the neck and shoulders is much more delicate than that of the thoracoscapular muscles so that an expansile pulsation can best be appreciated by placing the ear over the second right intercostal space. The diastolic shock can best be felt with the distal ends of the metacarpal bones.

Percussion reveals dullness in the second intercostal space to the right of the sternum. Accentuation of the second aortic sound is due not to an alteration of intra aortic pressure or a change in the structure of the valves but to the close approximation of the aorta to the chest wall. In the absence of aortic disease it occurs when the aorta is pulled to the right by fibroid disease of the right upper lobe of the lung. Aside from accentuation the second aortic sound may undergo a change in quality and become tympanic. This tympanic aortic second sound is indicative of disease of the aortic ring, the root of the aorta and possibly of the valves. It has nothing to do with accentuation and the occurrence of the two phenomena is quite distinct. Thus there may be accentuation alone or a tympanic quality alone or the two may be combined depending on the local lesion. When present the tympanic second aortic sound the *bruit de tabourka* of the French is of distinct diagnostic value. A systolic murmur in the second interspace to the right of the sternum always accompanies dilatation of the ascending aorta. It is produced by eddies in the blood stream as the blood passes from a vessel of smaller to one of larger lumen in this case as it passes through the normal aortic orifice into the dilated ascending aorta.

Roentgenoscopic examination is a valuable supplement to physical examination. Fluoroscopy yields more information than does the film. Roentgenoscopy will reveal only distal

tion and increased density of the aortic shadow, and these changes are common to aortic dilatation from various causes—atherosclerosis, hypertension, aortic insufficiency and syphilis. The information yielded by roentgen examination will rarely, by itself, allow of a positive diagnosis of syphilitic aortitis. It must be evaluated in connection with the other clinical data.

Dilatation of the ascending aorta in the anteroposterior view is manifested by an extension of the aortic shadow to the right of the sternum just above the arc formed by the right auricle. The aortic knob is more prominent on the left. The transverse diameter of the aortic shadow may be increased. Measurements of the width of the aortic shadow are of little value. When dilatation is extreme, measurements are unnecessary, when it is slight, their interpretation is equivocal. In the right oblique position, dilatation of the ascending aorta may be visible. In the left oblique position, in which the course of the whole arch is often visualized, the dilated first portion of the arch often stands out clearly on the left, arising just above, or even within, the shadow of the right auricle. Dilatation of the transverse and descending aorta is also readily demonstrable. An increased density of the aortic shadow may be apparent. This is due to the increased volume of the aorta filled with blood that must be penetrated by the roentgen rays and also to actual thickening of the aortic wall. Occasionally a sharply localized dilatation of the root of the aorta is seen. Such a finding is most characteristic of syphilitic aortitis. The other changes are found in other forms of aortic dilatation as well.

The diagnosis of syphilitic aortitis rests on the demonstration of the presence of dilatation of the aortic arch in a patient who is a known syphilitic. If there is accompanying hypertension or if the patient is over 60 years old, it is im-

possible to ascertain whether the dilatation is caused by a syphilitic lesion or by an atherosclerotic or simple dilatation. The younger the patient in the absence of complicating lesions the greater the probability that the dilatation is due to syphilis. A history of syphilis, a positive Wassermann reaction which occurs in about 75 per cent of cases or the finding of other stigmata of syphilis especially of cerebro-spinal syphilis are as important as the demonstration of the presence of aortic dilatation.

**Aortic insufficiency**—This is the most common complication of syphilitic aortitis and is found in over one third of the cases. It is usually a late manifestation of syphilitic infection of the aorta and when discovered, presages a relatively early death. Unfortunately

or so

The commonest complaint is dyspnea which is found as the presenting symptom in three quarters of the cases. It may take the form of ordinary dyspnea, such as is seen in sufferers from heart disease, appearing first after exertion and then gradually as compensation lessens becoming continuous or it may be paroxysmal. Dyspnea of either type is rather sudden in its onset.

Paroxysmal dyspnea is a dramatic and terrifying phenomenon. The attacks come suddenly, with little or no warning, and usually at night after the first or second hour of sleep. The patient awakens with intense air hunger and fear of suffocation. He may dash to the window to get more air or may evince marked motor restlessness.

When the most severe sort has set in the patient is usually placing his hands for additional support, and brings into use all of the accessory muscles of respiration.

Just as in true asthma, the dyspnea is ordinarily expiratory. The chest becomes hyperresonant, and squeaking râles and tracheal rhonchi become audible. The patient becomes pale and breaks into a cold sweat. As the attack continues, some cyanosis becomes evident in the face and extremities. Pain may or may not accompany the attack, it is not an essential feature. When it occurs it cannot be distinguished from the pain of angina pectoris. Quite constant, even in the absence of pain, are tremendous anxiety and fear of impending death. The pulse is rapid and the blood pressure usually greatly increased. I have not uncommonly seen figures of from 225 to 300 mm Hg systolic. There may be involuntary urination and defecation. The attack usually lasts from five to 30 minutes and subsides almost as suddenly as it begins. Toward the end of the attack the patient may cough up some blood-stained mucus. Pulmonary edema is not characteristic of these attacks but may supervene toward the end of prolonged severe seizures. After the attack, the breathing, pulse rate and blood pressure return to their original figures, and the patient, completely exhausted, sinks back on his pillows and often falls asleep.

The cause and nature of cardiac asthma, or paroxysmal dyspnea, have been much discussed during the past one hundred years, but we are still far from a complete answer. Hope,<sup>2</sup> writing in 1832, was the first clearly to distinguish the form of paroxysmal dyspnea that we have been discussing and to point out that it occurred particularly with hypertrophy and dilatation of the heart and aortic disease, that it was associated with little expectoration and that its incidence was chiefly nocturnal. It was Traube<sup>3</sup> who established the

<sup>2</sup> Hope, James A. *Treatise on the Diseases of the Heart and Great Vessels*

teaching which still prevails, that the asthmatic attack is caused by sudden failure of the left ventricle with resultant acute congestion of the lungs

Much of the lack of clarity that prevails is due to a confusion of different forms of severe dyspnea. The dyspnea seen in mitral disease, with acute edema of the lungs and signs of general venous stasis, is quite different from the paroxysmal form occurring in aortic disease. In the latter, all of the classic signs of congestive heart failure are absent. The paroxysmal dyspnea accompanying coronary disease, while resembling much more closely the form of cardiac asthma that we are discussing, differs in that signs of congestive heart failure quickly supervene and the blood pressure falls. An explanation of the paroxysmal dyspnea that is so characteristic in certain cases of marked left ventricular enlargement is by no means simple. Certainly some reflex mechanisms must be intimately concerned with the genesis of these attacks for they come and go too suddenly to be explained solely on the basis of acute failure of the left ventricle.

Wassermann<sup>4</sup> endeavored to reconcile all of these conflicting elements into a theory of cardiac asthma. The typical attack occurring during sleep is favored in its development by the physiological changes that accompany sleep. There is a preponderant vagus action, the heart slows, there is lessened oxygen consumption, with superficial respiration and a poorer aeration of the lungs. Many reflexes are abolished, and the threshold of response of the medullary centers is raised. Thus considerable anoxemia may develop before the usual compensatory adjustments are made. Suddenly, when it reaches a certain degree, all of the reflex mechanisms of defense seem to be activated, and many of the medullary centers respond

<sup>4</sup>Wassermann, S. *Neue klinische Gesichtspunkte zur Lehre vom Asthma cardiale* (Berlin: Urban & Schwarzenberg, 1926)

There appear the sudden violent respiratory movements, the bronchi are constricted through vagus action, the blood pressure is elevated, the pulse rate accelerated, the skin vessels constricted. There may be involuntary urination or defecation. The drug that gives greatest relief is not a cardiac stimulant but a depressant of the nervous system—morphine. As the attack continues, the increased blood flow, the excessive inspiratory suction on the blood in the large veins, together with the relative mitral insufficiency that is usually present, cause an overfilling of the pulmonary veins which may result in pulmonary edema. An analogous mechanism prevails in the diurnal attacks, but here the part played by failure of the left ventricle is greater, so that the added depressing effect of sleep is not essential to initiate an attack. From this it follows that diurnal attacks offer a graver prognosis than do nocturnal ones.

The appearance of paroxysmal dyspnea in a patient with syphilitic aortitis is always of serious import. It betokens a precarious state of the circulation. Patients may continue for months with these attacks and with a fair degree of comfort between paroxysms, but usually congestive heart failure soon supervenes, if death does not occur in the attack itself.

Pain occurs as an early symptom in two thirds of the cases of syphilitic aortic insufficiency. At times there is a constant dull pain under the upper portion of the sternum, frequently aggravated by exertion. More commonly the pain has the character of true angina pectoris. It may occur during attacks of paroxysmal dyspnea, or it may be associated with a rapid pulse rate and a rise in blood pressure. The occurrence of pain in these patients almost invariably points to encroachment of the syphilitic process on the mouths of the coronary arteries in the sinuses of Valsalva. These syndromes of pain, therefore, will be discussed in greater detail in the para-

graphis on coronary disease as a complication of syphilitic aortitis. Aortic insufficiency and the coronary syndrome are frequently associated.

Syphilitic involvement of the aortic valves gives rise to the most classic picture of free aortic insufficiency. There is no need to enter into the full details of the symptomatology and physical signs. It is often impossible to differentiate syphilitic from other forms of aortic insufficiency by the physical signs alone. Characteristically, there are great enlargement of the left ventricle, a loud gushing diastolic murmur to the left of the sternum or at the apex and a marked Corrigan pulse with all of the peripheral vascular signs. The pulse tracing of the subclavian artery has a sharp peak and never shows the anacrotic changes or the broadening of the peak often seen in rheumatic or atherosclerotic aortic insufficiency. The reason for this is that aortic stenosis, even of mild degree, which is so common in the latter two conditions, is never encountered in syphilitic aortic insufficiency.

Hypertension, with elevation of both systolic and diastolic pressures, occurs in about 15 per cent of the cases. I have seen figures as high as 260 mm. Hg systolic and 120 diastolic. In such cases granular kidneys are found at autopsy. The hypertension adds to the load on the left ventricle, hastens onset of heart failure and favors the occurrence of paroxysmal dyspnea.

Fever is a not uncommon feature of syphilitic aortitis and may provoke suspicion of an active rheumatic or bacterial infection. The differentiation may be difficult, particularly in view of the fact that moderate splenic enlargement is found in many patients with syphilitic aortitis.

Subacute bacterial endocarditis may arise from the engrafting of *Streptococcus viridans* on the syphilitic lesions. It is apt to take a rather virulent form and lead to early death.

It can be recognized by the many embolic phenomena, petechiae, red blood cells in the urine, infarcts of the spleen, kidneys or brain and by blood culture. Recognition of this complication in a patient with known syphilitic aortitis offers no difficulties. It is more difficult, when one sees for the first time a patient with manifest subacute bacterial endocarditis and an aortic lesion to determine whether syphilitic aortic insufficiency forms the original basis of the infection. A careful survey of all of the etiological and clinical features of the case will usually lead to the correct diagnosis.

Once syphilitic aortic insufficiency has become clinically manifest the progress of the disease is, as a rule, rapid. In general the more recent the syphilitic infection before the onset of cardiac symptoms, the more rapid the development of cardiac failure. The cases associated with *tabes dorsalis* are frequently indolent and asymptomatic and may go on for years. Indeed, in them the lesion is often discovered accidentally in the course of a routine examination. The average time between the onset of symptoms of syphilitic aortic insufficiency and death is given as about one year. This figure is based on hospital experience. It seems probable that in patients seen in private practice, who can take better care of themselves the average duration of life is much longer. The progress of the disease is marked by the development of signs of congestive heart failure. All of the chambers of the heart become greatly enlarged and the heart becomes progressively more and more insufficient. Congestion of the lungs and liver and peripheral edema set in. At this stage the heart does not respond to cardiac stimulants. Auricular fibrillation, however, rarely develops. This feature is important in differentiating syphilitic from other forms of aortic disease. The presence of auricular fibrillation even when the Wassermann reaction is positive, should arouse suspicion of another etiological factor.



Psychoses are common in the later stages of decompensation. As Head has pointed out, they occur more frequently in aortic disease than in any other form of cardiac defect, but they are not related to syphilitic affection of the brain. Hallucinations of vision and of hearing and a sense of anxiety appear. These are followed by paranoid delusions which may cause great motor excitement and restlessness. The appearance of a psychosis usually indicates an early lethal outcome.

Death results from progressive heart failure in about half of the cases of syphilitic aortic insufficiency. Sudden, unexpected death is another common mode of termination and is peculiarly characteristic of this valvular lesion. These sudden deaths are frequently due to coronary thrombosis, but often at autopsy no anatomical cause of death may be ascertained. One then predicates the onset of ventricular fibrillation. Not a few of these patients die of some intercurrent disease or of some complication such as embolism.

**Diagnosis.** The recognition of the syphilitic origin of a given case of aortic insufficiency is not always simple. The first and most important criterion is the establishment of the fact that the aortic valve is the only one diseased, for syphilis, in contradistinction to rheumatic and atherosclerotic processes, attacks none of the other valves of the heart. A history of syphilis and the absence of past rheumatic infection are important. The age of the patient is a valuable guide. Aortic insufficiency in individuals below the age of 30 is usually rheumatic, in those between the ages of 30 and 60 it is frequently syphilitic, in those over 60 it is usually atherosclerotic in origin. This rough rule applies in latitudes in which rheumatic fever is prevalent. Farther south, where rheumatic infection is rare, relatively more cases of aortic insufficiency are of syphilitic origin. A positive Wassermann reaction assists in the diagnosis, but from 10 to 20 per cent of persons with

syphilitic aortic insufficiency give negative reactions and positive reactions are not uncommon in individuals with heart disease and with syphilis which has not localized in the aorta. Syphilitic involvement of the central nervous system or of some other organ in the body may determine the diagnosis. Still more helpful is the presence of an aneurysm of the aorta. Aortic dilatation alone can rarely be used as a differential feature for in both rheumatic and atherosclerotic aortic insufficiency dilatation of considerable degree is common. Paroxysmal dyspnea and anginal pain while not pathognomonic occur much more frequently in the syphilitic forms of this valvular defect. Syphilitic aortic insufficiency never leads to aortic stenosis so that the presence of any signs pointing to aortic stenosis excludes syphilis as an etiologic factor. Similarly auricular fibrillation occurs so rarely in the syphilitic valvular defect that in its presence the diagnosis should be made with great caution.

*Coronary artery disease*—The syphilitic process in the aorta frequently encroaches on the orifices of the coronary arteries in the sinuses of Valsalva although the arteries themselves are rarely involved. This narrowing of the mouths of the coronary vessels restricts the flow of blood through them and gives rise to the syndrome of angina pectoris. Such coronary involvement may occur in simple syphilitic aortitis but is more common when the process has also extended to the aortic valves.

Pain occurring in patients with syphilitic aortitis is almost invariably due to narrowing of the coronary orifices. The pain does not differ from that encountered in the ordinary forms of angina pectoris but is more often associated with marked vasomotor reactions and more commonly is situated under the manubrium than under the midsternum. At first there may be just the usual substernal pain on effort with or

without radiation to the left shoulder and arm. In the classic form of angina pectoris, the patient is immobile during the attack, and there is no change of pulse rate, blood pressure or respiration. In many patients with syphilitic aortitis the anginal attacks are characterized by flushing of the face, marked acceleration of the heart, rise in blood pressure, increase in the respiratory rate and motor restlessness. Such attacks are seen as well in certain patients with hypertension and with rheumatic aortic insufficiency. It has been suggested that such attacks are caused not by coronary artery disease but by a reflex arising from the diseased aorta. These attacks should be differentiated from the more usual anginal attack. A number of such cases of angina have been described which at autopsy showed no disease of the coronary arteries, either along their course or at their mouths. Observations by Lewis support the view that, in such attacks, there is an actual spasm of the coronary arteries associated with a generalized vasoconstriction.

The anginal attacks are often but by no means invariably associated with seizures of paroxysmal dyspnea.

The diagnosis of coronary disease in patients with syphilitic aortitis is always suggested by the occurrence of angina pectoris. Further evidence may be found by electrocardiographic examination. Abnormalities in the T wave are the most frequent finding. Juster and Pardee, from a study of autopsy material, found that such abnormal T waves are due to encroachment on the lumen of the coronary orifices by the syphilitic disease in the sinuses of Valsalva. Such electrocardiographic changes were found in 85 per cent of patients with syphilitic aortic insufficiency and in only 38 per cent of those with uncomplicated syphilitic aortitis. The T wave may show the typical "coronary" form.

The prognosis is always grave in patients with syphilitic

aortic insufficiency and angina pectoris Sudden death is common

### TREATMENT

The only hope of curing syphilitic aortitis lies in treating the patient in the earliest stages of the disease, while the pathological process is still confined to the aorta proper But even with more advanced lesions, treatment intelligently carried out often leads to prolongation of life and relief from symptoms Carelessly given, it may hasten death or increase invalidity

A danger of antisyphilitic therapy lies in the Herxheimer reaction, a transient inflammatory response in the local lesion of the heart or aorta, which may lead to sudden death or acute congestive heart failure Rapid scarring of a gummatous lesion may induce rapid increase of the anatomical deformity in the heart Sudden death may follow directly on the administration of arsphenamine, due presumably to ventricular fibrillation Many cases are on record of patients who had never had congestive heart failure, who developed dyspnea, orthopnea, congested liver and edema directly following injudicious specific therapy Again, an aortic aneurysm may rupture within 24 hours of an injection More rarely, pre existing murmurs may become harsher or a fresh aortic diastolic murmur may develop during the course of treatment In these cases the scar accompanying healing increases the deformity of the valve

A growing appreciation of the complications that may follow antisyphilitic therapy, with a more hopeful outlook in regard to the results that may be achieved, has led in recent years to a safer, conservative plan of treatment which, in expert hands, has given good results

Certain patients with syphilitic aortitis are best left un-

treated. These are patients without symptoms, usually individuals 65 years or older, often with neurosyphilis as well, in whom the cardiovascular lesion has been discovered accidentally. In them the prognosis, even if they are left alone, is fair, and treatment may in some way disturb the balance between the activity of the spirochetes and the resistance of the body and lead to rapid progress of the lesion. The Wassermann reaction of the blood is no guide to treatment. Moreover, as Moore has pointed out, clinical and pathological progress of the lesion may occur in the presence of a negative Wassermann reaction of the blood.

In all cases, specific treatment should be delayed until signs of circulatory failure and the subjective symptoms of pain or paroxysmal dyspnea have been largely relieved by general medical treatment. This includes bed rest, limitation of fluids and salt and the administration of digitalis and diuretics.

When the symptoms of cardiac failure have been controlled, antisyphilitic therapy may be carefully instituted. It is best to begin with the intramuscular injection of bismuth in oil, 0.1 Gm. every four to five days. If this is well tolerated, the dose is increased to 0.2 Gm. after five injections, and this is given once a week. Potassium or sodium iodide is given by mouth at the same time as the bismuth in a dosage of 1 Gm. three times a day, increasing rapidly to 4 Gm. three times a day. This treatment with bismuth and iodides is continued for 12 weeks. If the patient has not recovered a satisfactory cardiac reserve, no arsenical is given, but the same course of treatment is repeated after a rest period of about two months. If there is no cardiac embarrassment, the bismuth injection is followed by a course of neoarsphenamine. Arsphenamine itself should not be used for it more readily produces untoward reactions. The initial dose of neoarsphenamine is

0.1 Gm., and this is cautiously increased at weekly intervals until a maximum dosage of from 0.3 to 0.4 Gm. is reached. Twelve injections are given. Then the course of bismuth and iodides is resumed, to be followed again by neoarsphenamine. Mapharsen is less toxic than neoarsphenamine and may be used in its place. The initial dose is 0.02 Gm. It should be gradually increased to a maximum of 0.04 Gm. Ideally, treatment should be continuous for at least two years, and even after this period has elapsed it should be continued at intervals as long as the patient lives. Mercury injections may be substituted for bismuth, if desired.

Penicillin will probably displace the arsenicals in the near future, but the effective dosage and course of treatment have not yet been worked out. Adequate treatment probably requires 80,000 units every three hours day and night for 7½ days. This should be preceded by a course of bismuth.

Untreated patients with syphilis of the aorta die, on the average, in 12 to 24 months after the onset of symptoms. Moore, in an exhaustive study of 169 patients, showed that in well treated patients the average duration of life can be extended to about six years. Many of these patients are relieved of pain and dyspnea, remain free from cardiac symptoms and resume work.

The treatment of paroxysmal dyspnea and of angina pectoris due to syphilitic aortitis is the same as that outlined for patients in whom these syndromes result from coronary artery disease.

In patients in whom severe anginal seizures persist in spite of all therapy, paravertebral nerve block with alcohol is very useful and often gives complete relief. The same procedure may be used in patients in whom pressure or erosion of a large aneurysm causes much pain. The ganglia to be injected are determined by the distribution of the pain.

*THE PATIENT PAST FIFTY*

In rare cases of saccular aortic aneurysms the attempt may be made to cause a clot to form in the aneurysmal sac by inserting many yards of fine wire into the sac and passing a galvanic current through the wire. This method, or one of its variations, has been employed with varying success by many surgeons.<sup>5</sup> It can be used only in saccular aneurysms with a small communication with the aorta. At best, this treatment results in complete closure and fibrosis of the aneurysmal sac, but failures are more frequent than successes, and complications such as embolism, hemorrhage or infection are not uncommon. The many other methods that have been employed to obliterate the aneurysmal sac are still less successful.

J. M. T. "Wiring of Otherwise Inoperable Aneurysms" *Am.*  
661, 1912.

## *Chapter Eight*

# **DISEASES OF THE CARDIOVASCULAR SYSTEM— VALVULAR DISEASE AND IRREGULARITIES**

ALTHOUGH coronary artery disease and hypertension are responsible for the majority of cases of heart disease in persons past the age of 50, valvular heart disease is by no means uncommon. Syphilitic heart disease which leads to aortic insufficiency has been discussed in Chapter 7. It accounts for about 10 per cent of all cases of heart disease in adults, but a somewhat lower percentage in the older age periods.

Although rheumatic fever and rheumatic valvular disease are preeminently conditions encountered in the young, many cases are seen in older persons. As a rule, rheumatic fever with consequent damage to the heart valves attacks persons under the age of 20. The majority of these die of rheumatic reinfection or of the mechanical effects of their valvular defects before they reach their fortieth year. Some 10 per cent of those who contract rheumatic heart disease in their youth survive to the age of 50. These are persons who have had a relatively mild infection, with few or no recurrences and with little mechanical handicap to the heart. Among older persons with rheumatic valvular disease there are also those who contracted their initial rheumatic infection at a more advanced age—in the fourth or fifth decade. Rarely, the initial



rheumatic infection occurs after the age of 50. Rheumatic valvular disease occurs, but is unusual, after the age of 60. In most of these cases the valvular deformity and the resulting mechanical handicap to the heart are not extreme and the lesion is nonprogressive, or very slowly progressive, so that significant symptoms do not develop for many years. Often in later life an atherosclerotic process with calcification is superimposed on the valve scarred by the ancient rheumatic infection, and it is this secondary atherosclerosis that, by further damaging the valve, leads to heart failure. Even in elderly persons rheumatic reinfection occurs, and many instances of heart failure in older persons with rheumatic valvular disease are due to acute or subacute rheumatic carditis. Rheumatic valvular disease may affect the mitral or aortic valves, or both. In the aged tricuspid disease is unusual. With mitral disease, auricular fibrillation is common.

Arteriosclerotic valvular disease occurs after the age of 60. It involves the aortic valve more often than the mitral.

### MITRAL STENOSIS

Mitral stenosis is not uncommon between the ages of 50 and 60. About 20 per cent of all cases of mitral stenosis are encountered in persons past the age of 50. Most of these are rheumatic in origin, and in some the lesion dates to a solitary mild rheumatic infection in childhood. I saw a man of 64 whose mitral lesion was due to a solitary attack of polyarthritis at the age of 7. After the age of 60, one encounters occasional cases of arteriosclerotic mitral stenosis. The lesion in these cases is a nodular calcification of the fibrous auriculoventricular ring, which narrows the auriculoventricular orifice, but to a less degree than does the typical endocarditic form of mitral stenosis. The mitral leaflets are thickened and

sclerotic but not fused. The calcified ring may be visualized by fluoroscopic study of the heart, if the observer is well accommodated. The consequences of mitral stenosis in elderly persons are identical with those in the young. First, there is enlargement of the left auricle and right ventricle, followed later by left ventricular enlargement. Eventually, heart failure results. Auricular fibrillation is a common complication. Hypertension occurs in fully one half of all persons with mitral stenosis past the age of 50. The cause for this association is not clear. Embolism to the cerebral, renal or peripheral arteries is common. The embolus arises from a thrombus in the left auricle.

#### AORTIC INSUFFICIENCY

Aortic insufficiency, when it is associated with mitral stenosis, is due to rheumatic infection. Solitary aortic insufficiency in older persons may be due to syphilis, to arteriosclerosis or to a functional dilatation of the aortic ring. This last condition arises only with marked hypertension or with grave anemia. After the age of 60, arteriosclerosis is the most common cause of aortic valvular disease. Syphilitic aortic disease has been discussed in Chapter 7. The diagnosis of rheumatic aortic insufficiency depends on a history of rheumatism and on associated mitral disease. Arteriosclerotic aortic insufficiency is diagnosed by eliminating other possible causes of the lesion. If there is associated aortic stenosis, syphilis can be safely excluded. Traumatic rupture of a semilunar cusp is a rare cause of aortic insufficiency and occurs only in a valve previously weakened by disease. Aortic insufficiency is followed by marked enlargement of the left ventricle and eventually by left ventricular failure. The rheumatic and syphilitic cases show the usual peripheral manifestations of

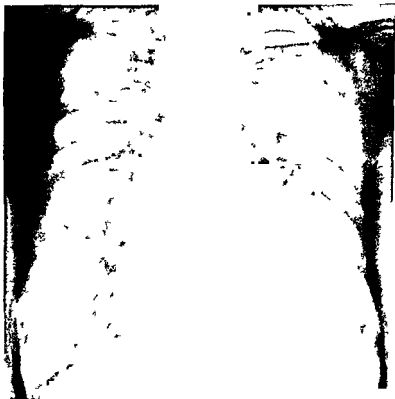


FIG. 14—Chronic rheumatic valvular disease with aortic insufficiency  
Note great enlargement of the heart especially of the left ventricle

aortic insufficiency water hammer pulse, high pulse pressure and so called capillary pulse Arteriosclerotic aortic insufficiency and functional aortic insufficiency are less apt to exhibit these changes in the peripheral circulation Aortic insufficiency due to atherosclerosis adds little to the gravity of the underlying disease, and does not appreciably alter the clinical picture These patients are usually suffering from arterial hypertension or from atherosclerosis of the aorta, and their future depends more on the progress of these conditions

### AORTIC STENOSIS

Calcareous aortic stenosis may result from calcareous deposits in an old rheumatic valvular lesion, or it may arise as a primary arteriosclerotic process Rheumatic aortic stenosis is usually associated with a mitral lesion, there is often history of ancient rheumatic infection Arteriosclerotic aortic stenosis occurs in persons past 60, and chiefly in males it is a solitary valve lesion, and there is no history of antecedent rheumatic infection Aortic stenosis does not result from syphilitic infection

The physical signs of rheumatic and arteriosclerotic aortic stenosis are identical A rough systolic murmur, usually accompanied by a systolic thrill, is heard and felt at the aortic area In the presence of heart failure the murmur may become soft and faint and the thrill may disappear Aortic stenosis presents symptoms different from those of other valvular lesions The syndrome of angina pectoris is common It is caused not by disease of the coronary arteries but by narrowing of the aortic valvular opening itself, which impairs the blood supply to both coronary arteries simultaneously The presence of aortic stenosis should be suspected when a patient who for years has had a heart murmur or a valvular

lesion subsequently develops angina pectoris. Sudden heart failure in these patients induces coronary insufficiency and is often followed by symptoms simulating coronary thrombosis. Partial and complete heart block are also often associated with calcareous aortic stenosis. These are caused by extension of the calcific process into the ventricular septum with involvement of the auriculoventricular bundle. Sudden syncope is common in patients with aortic stenosis and death often comes instantaneously and unexpectedly. The diagnosis of calcareous aortic stenosis can in a large number of instances be confirmed by visualizing the calcified aortic ring fluoroscopically when the observer's eyes have been fully accommodated.

Treatment of patients with chronic valvular disease does not differ from that of other patients with heart disease. The patient should keep his activities within the range set by his cardiac reserve and this is usually signaled by the appearance of dyspnea when he overtakes himself. Digitalis is given only when there is heart failure. With the onset of heart failure the patient should be put to bed and treated by the usual measures. Even in older persons with rheumatic valvular disease heart failure may be induced by rheumatic carditis accompanying a rheumatic reinfection. Arthritis is rare and the temperature does not range high so the infection is difficult to recognize. Its presence may be suspected when the patient does not respond to treatment.

### CARDIAC IRREGULARITIES

*Extrasystoles* or premature beats are very common in older individuals; they are usually ventricular in origin. They may occur in hearts that are otherwise normal or may appear in hearts that are diseased. As a rule they are of no clinical

significance. They may result from tobacco, coffee or digitalis intoxication.

Their sudden appearance accompanying an acute infectious disease, such as pneumonia or erysipelas, gives evidence that the heart has been damaged by the toxins or virus of the disease. Auricular extrasystoles in patients with great cardiac enlargement are usually forerunners of auricular fibrillation. Numerous extrasystoles occurring in patients with arteriosclerosis of the coronary arteries, particularly when associated with anginal attacks, usually indicate a progressive vascular lesion. Short runs of extrasystoles, or multifocal extrasystoles in such patients at times may act as a trigger mechanism initiating ventricular fibrillation which is followed by sudden death.

Extrasystoles, as such, require no treatment unless they disturb the patient or unless there is reason to fear the development of ventricular tachycardia or fibrillation. It is the underlying heart condition that determines the treatment. In the absence of demonstrable heart disease, extrasystoles may be ignored. If it is desirable to stop the irregularity, this can usually be achieved by giving quinidine sulfate in doses of 3 gr. three or four times a day. At times digitalis,  $1\frac{1}{2}$  gr. twice a day, may succeed when quinidine fails.

*Auricular fibrillation* occurs in older patients as it does in the young in long standing heart disease with considerable cardiac enlargement, particularly enlargement of the left auricle. It is seen in patients with rheumatic, hypertensive or arteriosclerotic heart disease. Auricular fibrillation may appear suddenly as a complication of cardiac infarction. In such cases there is often a spontaneous return to sinus rhythm. As in younger patients auricular fibrillation occurs with hyperthyroidism, and in elderly persons it may be the presenting sign of the thyroid disorder. Finally, there are cases in which

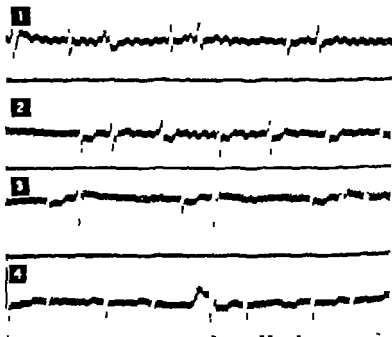


FIG 15—Electrocardiogram Auricular fibrillation in a patient without demonstrable organic heart disease

auricular fibrillation is the sole evidence of cardiac abnormality. It is not uncommon in older persons and has been called arteriosclerotic auricular fibrillation. It is doubtful whether this designation is correct, for there may be no gross evidence of arteriosclerosis and the condition is also encountered in younger individuals. In the elderly auricular fibrillation without demonstrable associated heart disease causes little disturbance. The ventricular rate is usually slow and there is little dyspnea or heart failure. In itself it does not shorten life nor does it greatly impair the efficiency of the individual.

The treatment of auricular fibrillation is the treatment of the underlying heart condition. Digitalis should always be given in dosage sufficient to slow the ventricular rate to between 70 and 80 and to keep it at that level. When the heart rate is rapid and there is much dyspnea and congestive failure large doses may be given at first—4 cat units twice a day for two days. After this the dose should be adjusted to meet the needs of the patient. Usually 1 or 2 cat units daily is the maintenance dose. The medication must be continued indefinitely as long as the irregularity persists. In elderly persons the heart rate can be controlled by small doses of the drug. Often 1 cat unit every day or every other day is sufficient.

*Heart block* complete or partial in elderly individuals is due almost invariably to myocardial fibrosis consequent to coronary artery disease. Sudden development of heart block is often associated with infarction of the posterior wall of the left ventricle for with this lesion the blood supply to the auriculoventricular node may be affected. Such heart block may be permanent or may recede in a few days. Treatment is that of the underlying heart condition. In the presence of partial heart block digitalis should be used with great caution or not at all. It may convert partial into complete block. In the presence of complete block digitalis may be used



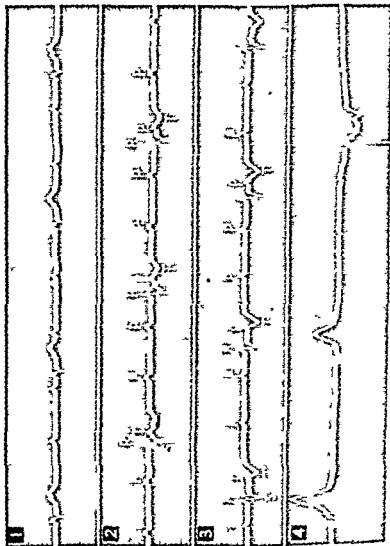


Fig. 10—Electrocardiogram showing complete heart block.



FIG 17 -Electrocardiogram Bundle branch block

freely to combat heart failure. At times attacks of Stokes Adams syncope occur in these patients. There is sudden cessation of the heart sounds, the patient becomes unconscious and may have a convulsive seizure. After periods of time varying from five to 60 seconds the heart resumes its beating and consciousness returns. The Stokes Adams syndrome in some cases is due to ventricular standstill and in others to transient ventricular fibrillation. It is important to try to differentiate between the two by taking an electrocardiogram during the attack. If there is transient ventricular fibrillation the administration of quinidine sulfate 3 to 5 gr every four hours should stop the seizures. In these cases adrenalin is dangerous for it may induce or favor ventricular fibrillation. If the attack is due to ventricular standstill quinidine is useless. Hypodermic injection of adrenalin 5 to 10 minims usually arrests the attack. The dosage should be continued at appropriate intervals depending on the frequency of the attacks. Ephedrine hydrochloride  $\frac{1}{2}$  gr four times a day is often effective. If these drugs fail one may try barium chloride  $\frac{1}{2}$  gr three times a day. With careful treatment the attacks of syncope can often be controlled. Often the attacks are initiated by acute myocardial damage and if the patient can be tided over the acute period complete recovery may ensue.

### BACTERIAL ENDOCARDITIS

Bacterial endocarditis both the acute and the subacute form is encountered with sufficient frequency among older persons to oblige the physician to suspect its presence in every case of obscure fever.<sup>1</sup> Approximately 10 per cent of

<sup>1</sup>Zeman F D and Segal S "Acute Bacterial Endocarditis in the Aged" *Am Heart J* 29:597 1945. Zeman F D "Subacute Bacterial Endocarditis in the Aged" *ibid* p 691 1945.

all cases of subacute bacterial endocarditis occur in patients over age 50

*Acute bacterial endocarditis* is induced by one of the pyogenic bacteria that gains admission to the blood stream through some suppurative focus, such as a pneumonia, a breaking down carcinoma, an infected kidney or mastoid or a carbuncle of the skin. The presence of diabetes often accelerates such infection, and the existence of a heart valve damaged by an ancient rheumatic infection favors the lodgment of bacteria. Vegetations may develop on healthy valves. The pneumococcus is one of the organisms most frequently encountered in acute bacterial endocarditis in older persons. As a rule the primary infection in the lungs, kidneys or elsewhere dominates the clinical picture and the diagnosis is overlooked. Examination of the heart may reveal only a systolic murmur, the significance of which is difficult to evaluate in older persons. Petechiae or other emboli or an enlarged spleen may suggest the presence of endocarditis. The diagnosis will be made only if blood cultures are made in the presence of sepsis particularly when associated with chills. Positive results of a blood culture usually indicate bacterial endocarditis.

Intensive administration of penicillin as outlined in the following section on subacute bacterial endocarditis, offers some hope of cure. At the same time attempts should be made to drain or eliminate the primary focus of infection.

*Subacute bacterial endocarditis* is caused by infection with *Streptococcus viridans*, *enterococcus* or *Hemophilus influenzae*. With rare exceptions these organisms induce inflammatory vegetations only on valves damaged by previous disease. Even in elderly persons the most common antecedent lesion is rheumatic valvular disease although infection may occur on syphilitic or arteriosclerotic valves and in rare cases on a

congenital anomaly with which the patient has survived to an advanced age. The clinical picture commonly resembles that seen in younger persons. A patient with a valvular defect has fever, possibly chills, embolic phenomena such as petechiae, red blood cells in the urine, infarcts of the spleen, kidney or brain, enlargement of the spleen, clubbing of the fingers. Blood culture reveals the infecting organism. In such typical cases the diagnosis is simple if the clinician will recall that the disease occurs in older persons. The clinical picture may be more obscure. Low grade fever may be the only presenting sign. Murmurs in old people are always difficult of interpretation and are too apt to be dismissed as being due to arteriosclerosis of the valves. Heart failure may overshadow the other symptomatology. Only a positive blood culture makes the diagnosis definitive. If the blood culture at first is negative it should be repeated several times, for often the organism grows with difficulty on artificial mediums.

Healed subacute bacterial endocarditis, a condition in which there have been spontaneous healing and sterilization of the valvular vegetations, presents a different clinical picture. The patient has a darkly pigmented skin, there may or may not be fever with occasional emboli. There are splenomegaly, renal insufficiency with azotemia and progressive anemia. Blood cultures remain negative.

**Treatment.** With penicillin some 75 per cent of patients with subacute bacterial endocarditis can be cured of their infection. Some die of embolism or heart failure. Dosage of the drug should be carefully planned; for insufficient initial dosage may make the infecting organism resistant to penicillin and so prevent a cure. Inadequate total dosage temporarily arrests the infection but permits a recurrence when treatment is stopped. The first step is to determine the degree of resistance to penicillin of the infecting organism. The dos-

age is adjusted to the coefficient of resistance as set forth in Table 5

TABLE 5—DOSE OF PENICILLIN FOR ORGANISMS WITH VARYING COEFFICIENTS OF RESISTANCE\*

COEFFICIENT OF RESISTANCE OF ORGANISM	DOSE BY INTRAMUSCULAR INJECTION EVERY 3 HRS	PEAK BLOOD CONCENTRATION TO BE ACHIEVED IN UNITS CC BLOOD SERUM
1	15 000	0.5
2-3	25 000	1.0
4-5	50 000	2.0
5-10	100 000	6.0
10-20	200 000	11.0
20-30	300 000	16.0

\* From Gerbe, I. E. et al. Penetration of Penicillin into Focus of Infection. J. A. M. A. 130:761, 1946.

It is well to give additional doses of from 50 000 to 100 000 units twice a day. The whole purpose is to maintain blood levels of penicillin that will destroy the infecting organism. Such large doses bring about peaks of high penicillin blood levels. Intramuscular administration is more effective than intravenous injection. Treatment should be continued for at least four weeks. Usually the fever and the active symptoms are controlled within a few days, but prolonged treatment is essential to make certain a permanent cure.

Organisms that are more than 30 times as resistant as the standard strain of staphylococcus rarely can be eliminated by treatment with penicillin. In such cases streptomycin may be successful. I have seen several patients with highly resistant enterococcus infection cured with streptomycin.

## *Chapter Nine*

# **DISEASES OF THE CARDIOVASCULAR SYSTEM-- GENERAL MANAGEMENT**

THE TREATMENT of the patient with a diseased heart is not a simple matter. It involves more than knowing when and how to administer digitals. It demands, first of all, an accurate and complete etiological, physiological and anatomical diagnosis. It calls for understanding and patience to grasp the patient's many problems and to help him readjust his whole life to the functional capacity of his damaged heart. It predicates a comprehension of the unique relationship between heart and psyche through the intermediation of the vegetative nervous system. Finally, it demands an unwavering faith in the efficacy of treatment and in the astounding recuperative power of the heart. So often, by calling on its last ounce of reserve, does the failing heart re-establish the circulation and give the patient a fresh hold on life that there is hardly ever justification for the verdict "This case is hopeless." The admission of defeat encourages defeat.

We shall first discuss the management of patients with arrested or slowly progressing heart lesions that do not limit physical activity or at most give rise to slight symptoms on greater activity.

The patient should be told that he has a cardiac abnor-

malilty, but he must be made to understand that the lesion does not endanger life, that it merely calls for reasonable caution. The heart is quite able to meet the demands made by the ordinary exigencies of life but has not the reserve power to withstand greater and extraordinary drains upon its strength. In perfect health the heart can take care of all the needs of the body. In the presence of a damaged heart, body activity should be adapted to the cardiac capacity, indeed should be kept at a level a bit below that capacity. The measure of the strength of the heart is its response to effort. Heart strain is signilized by the subjective symptoms of dyspnea, palpitation and precordial pain. The amount of effort required to provoke these symptoms is a gage of cardiac reserve power.

But dyspnea, palpitation and precordial pain may occur also with functional neurogenic disturbances of the circulation, obesity or severe anemia. To be indicative of early heart failure, these symptoms must be associated with a cardiac lesion capable of provoking them or with physiological disturbances of the heart such as alterations in rate or rhythm. When a neurogenic cardiovascular disturbance is superimposed upon an organic lesion, the interpretation of the symptoms may be difficult. Women with hypertension often complain of hot flushes, vertigo, rapid palpitation, dyspnea and sticking precordial pain, determined by a neurogenic disorder.

In such patients the symptoms are out of all proportion in their intensity to the anatomical changes in the heart. Were they determined by heart failure, one would find a large dilated heart, distended neck veins, cyanosis and congestion of the lungs or liver. Instead, the heart is small and there is no venous congestion. The symptoms must always be correlated with the heart lesion. In patients with psychoneurotic disturbances manifesting themselves in a cardio-



vascular symptomatology, the erroneous diagnosis of heart failure and the restrictions and treatment consequent thereon may do incalculable harm.

Thus the first task of the physician is to enlist the patient's aid in working out his exercise tolerance. This is best achieved by studying his response to his regular daily mode of life. If the patient can carry on without any symptoms, his cardiac reserve is excellent and his activities need not be curtailed. If certain efforts, such as climbing stairs, carrying heavy parcels or sweeping, give rise to discomfort or induce undue fatigue or if the patient is exhausted in the evening after his round of duties, he is overstraining himself and his heart. In many instances, merely slowing the tempo of activity enables the individual to carry on without distress. The patient must learn to regulate the amount and vigor of his movements to a point below that which will give rise to symptoms. Thus, if climbing two flights of stairs brings on shortness of breath, he should, if he must ascend two stories, rest after the first flight so that the second one may be negotiated without discomfort.

### CLIMATE AND ALTITUDE

*Climate*—This has little direct influence on the progress of heart disease, and there are no general rules applicable to all patients. Insofar as climate contributes to respiratory infections it is of great importance, for rheumatic recrudescences are often initiated by such infections, and in all forms of heart disease the load placed on the circulation by an attack of bronchitis or bronchiopneumonia may precipitate heart failure. This last complication is to be feared particularly in patients with advanced pulmonary emphysema. The winters in the Middle Atlantic states and in New England, with their great variations in temperature and moisture, are a real

hardship to individuals with damaged hearts. A warm, equable climate such as is found in the South is to be preferred, indeed, it is almost mandatory for those with more advanced lesions who are particularly susceptible to colds and respiratory infections. If there has been no heart failure, the necessity for a change of climate is not so exigent. It is usually preferable to permit the patient to continue the normal routine of his life rather than subject it to such violent dislocation as is usually entailed by a winter move to the South. If, in addition to the cardiac affection, there is a chronic sinus infection, the moist climate of the southern seaboard may be unfavorable. In such cases the dry warmth of Arizona or New Mexico may be more beneficial. Patients with angina pectoris always have greater difficulty in cold windy weather. Many feel quite well during the summer but when winter comes find their activities greatly restricted because of the frequent occurrence of anginal attacks in cold blustery weather. For them a warm climate is very helpful and comforting.

There are some who do not tolerate heat, particularly women with hypertension. They experience flushes, headaches, palpitation and increased weakness at higher temperatures. Florida climate does not agree with them.

*Altitude*—The rarefied atmosphere at high altitudes causes anoxemia which may be of sufficient degree to burden the heart. The chief evidence of such heart strain is an increased pulse rate, markedly exaggerated by slight exertion. Cardiac dilatation may ensue. Such effects are produced at altitudes of 14 000 ft. or more, rarely at lower levels.

Patients with heart disease have a fear of altitude, and their physicians often counsel them against spending a summer in the mountains. Altitudes up to 6,000 or 7,000 ft. have no effect upon the heart. Indeed, some claim that such heights benefit certain forms of heart disease. Sojourns in mountain-

ous country rarely injure subjects of heart disease because of the altitude as such, but rather because the nature of the terrain may compel them to do too much climbing

The patient with organic heart disease need not avoid moderate heights such as are found in the Adirondacks, the Catskills or the Appalachians, but he should avoid undue climbing which, even at lower levels, may strain his heart. The choice of a summer resort or of a permanent home need not be dictated by the condition of the heart.

Airplane flights not exceeding 10,000 ft in height are not injurious to the heart by reason of the high altitude. The psychic influence of the excitement of taking off or the effects of air sickness may far outbalance any possible effect induced by the rarefied atmosphere. Whether a patient with heart disease may undertake an airplane flight depends chiefly on his psychic make-up. The stratosphere flights of the future will probably be taboo.

#### ANESTHESIA AND OPERATION

Contemplation of operative intervention in a patient whose heart is diseased calls for an evaluation of the added risks that the cardiac condition introduces into the surgical procedure. Misjudgment results when heart disease is regarded as a unitary disorder. A variety of diseases affect the heart, and their effects on cardiac function differ at different periods of their evolution. Clarity of appraisal is reached with the recognition that there is no one form of heart disease but that there are heart diseases, as well as hearts with different degrees of functional capacity.

The cardiac disease hardly increases the operative mortality in patients who have no history or evidence of heart failure but have chronic rheumatic valvular disease, uncom-

plicated essential hypertension or heart disease due to hyperthyroidism or congenital defects. For this group of patients, the criteria for anesthesia and operation are practically the same as for normal individuals. The operative mortality hardly exceeds that of normal individuals. They may be subjected to emergency operation without fear. Operations of choice, such as herniotomies and cholecystectomies may be carried out. There is much to be said for operating early while the heart is still well compensated. Delaying until the condition has become advanced may necessitate operation at a time when the heart has begun to fail. In these patients there is no need for the pre- or postoperative administration of digitalis. Fluids may be given freely both by mouth and intravenously.

Patients with coronary artery sclerosis and with syphilis of the aorta and aortic valves are less favorable subjects for surgery. A recent study revealed an unexpected operative mortality of 8 per cent. All but urgent operations should be avoided. The expectancy of life of these patients is short and the need for operation should be balanced against the prognosis for the heart condition. If operation becomes necessary, a short anesthesia, speedy operation and minimal trauma and bleeding must be assured. In these patients an unforeseen coronary thrombosis may develop at any time even as late as several weeks after the operation. The frequency of this association is such as to render a causal connection probable. Patients with these forms of heart disease are subject to sudden death due presumably to ventricular fibrillation. This seems to occur more frequently when there is a pre-existing extrasystolic irregularity. To such patients it may be wise to give 3 gr. of quinidine sulfate three or four times a day for several weeks.

For patients with congestive heart failure, operation is

hazardous and should be resorted to only as a life saving measure. These patients show an unexpected mortality of 17 per cent. Here is need for a surgeon of real virtuosity. There is little time for preoperative preparation of the patient. The most important measure is the speedy administration of full doses of digitalis. If the patient has had no previous medication with digitalis he may be given 1.2 mg of digitoxin (Digitaline Nativelle or Purodigin) by mouth. This is the full digitalizing dose. The maintenance dose is 0.2 mg a day. Or else 20 gr or 15 cat units of digitalis leaf may be given in divided doses in 24 hours. The maintenance dose is from 1 to 2 cat units a day. If there is much venous congestion a phlebotomy of 300 to 500 cc may be helpful. In these patients the administration of fluids intravenously may place too great a strain on the heart and may even precipitate an attack of pulmonary edema. This happens more often with the transfusion of blood than with the infusion of saline. When it becomes necessary to administer fluids intravenously or to give a transfusion the injection should be given slowly by the drip method and the patient should be kept under constant observation. Of the supplementary cardiac stimulants caffeine sodium benzoate 7.5 gr given every three to four hours is best. Restlessness should be combated with morphine and appropriate sedatives. In the presence of much dyspnea and cyanosis oxygen quiets the patient and eases the work of the heart.

The presence of auricular fibrillation as such does not rate is  
 scope—between 70 and 80 beats a minute. Measurement of  
 the pulse rate at the wrist is inaccurate because of the com-  
 mon presence of a pulse deficit. Auricular fibrillation as a

rule, develops in the late stages of heart disease, so that it occurs in the more badly damaged hearts, except in patients with hyperthyroidism

When circulatory embarrassment is induced or exaggerated by a condition amenable to surgical treatment, operation is imperative. It offers the only chance of relieving the heart of its extra burden and of restoring compensation. The commonest condition of this type is hyperthyroidism, which has either directly led to the heart disease or has aggravated some form of preexisting heart disease. Congestive heart failure is seen only in cases that persist over many years. It is rather uncommon and occurs almost exclusively in patients who have coexistent organic heart disease.

At times a damaged heart is further weakened by the presence of severe secondary anemia occasioned by bleeding fibroids, hemorrhoids or some analogous condition. In these cases the anemia as such contributes to the heart failure, so that there is definite indication for operation to arrest bleeding. Preoperative treatment with bed rest, digitalis, diuretics and, particularly, transfusion to restore the normal oxygen-carrying capacity of the blood will bring about sufficient improvement to make operation possible. Transfusions of citrated blood may be given—very slowly by the drip method—even in the presence of heart failure. It is wiser to give small quantities, 250 cc daily, rather than to attempt to administer 500 cc at a time.

In all operations on patients with heart disease, pre- and postoperative care must be meticulous, particular attention should be given to the prevention of preoperative anxiety and to cardiac strain incident to the excitement stage of anesthesia. A highly skilled anesthetist is always required. In selected cases local anesthesia may be best, in many, avertin (tribromethanol) is most satisfactory as a basal anesthetic.

Chloroform, which is little used in this country, is best avoided because of its known tendency to induce ventricular fibrillation. Spinal anesthesia, as a rule, is not adapted to cardiac patients because of the drop in blood pressure that so often ensues. This is particularly marked in individuals with arterial hypertension and in them spinal anesthesia should be avoided. Most patients with cardiac disease tolerate ether well, and in most instances it is the best anesthetic. In the presence of emphysema and bronchitis, ethylene or cyclopropane is better borne than ether.

Often the nervous tension incident to anticipation of the operation quickens the heart rate to a level higher than that provoked by the operation itself. This occurs particularly when the patient is brought into the operating room fully conscious and the anesthesia is started with the patient on the operating table. Again the excitement stage of the anesthesia, whether it be nitrous oxide, ether or ethylene, is always accompanied by a great rise in heart rate which usually exceeds the maximum rate during operation as well as that induced by preoperative excitement. Fluctuations in rate are closely linked to the depth of anesthesia, when the anesthesia becomes light, the heart rate quickens. In a placid individual, local anesthesia is often associated with a remarkable stability of heart rate. In avertin anesthesia the excitement stage with rapid pulse is usually avoided. During the operation itself the heart rate is usually remarkably constant and is little influenced by manipulations of tissues or viscera unless great hemorrhage or shock sets in. Anesthesia may cause abnormalities of cardiac mechanism such as extrasystoles or paroxysmal auricular tachycardia. These are of little clinical significance, for they almost invariably subside spontaneously.

## HEART FAILURE

The treatment of heart failure is the same, no matter what the age of the patient. Symptoms of heart failure, whether they be dyspnea, cough, edema or chest pain, are evidence that the heart is overtaxed. The first principle is to relieve the heart of all unnecessary strain. To rest the heart one must rest the body, so the first indication is to put the patient to bed. Bed rest should be continued until the evidences of heart failure have subsided. Digestion, just like bodily activity, increases the work of the heart so the diet should be kept light and scanty.

In chronic heart disease with recurring heart failure, the patient's appetite is poor and he is apt to suffer from chronic undernutrition. His protein intake has usually been low for weeks or months, often to such an extent that the level of blood proteins is reduced. In the absence of complicating renal disease, very low figures are not reached, but levels of 5.5 to 6 Gm per 100 cc of blood are common. This lowered blood protein level favors the development of edema, particularly with the rise in venous pressure that accompanies heart failure. It is well, therefore, to prescribe a diet high in protein. Often the administration of thiamine hydrochloride, 3 mg twice a day, and nicotinic acid, 50 mg three times a day, seems to be helpful.

In all cases of heart failure efforts should be made to control abdominal distention which, by pushing up the diaphragm, may embarrass the heart. With the onset of heart failure, urinary excretion diminishes and there is retention of fluid in the body which may eventually cause an increase in blood volume and thus increase the work of the heart. Control of the fluid exchange of the body is fundamental to the treatment of heart failure.



The fluid intake should be restricted to 1,000 cc. in 24 hours. The fluid intake and output should be recorded.

With rest and fluid restriction, heart failure subsides and compensation is restored. This can be hastened by the administration of digitalis. In mild cases 1 cat unit of digitalis or 0.2 mg. of digitoxin twice a day is sufficient. If heart failure is severe and if the patient has had no digitalis medication, he may be digitalized rapidly. The average full digitalizing dose is 20 cat units of the leaf or 1.2 mg. of digitoxin. This is only a rough guide, for there is great individual variation among patients in the doses of digitalis that they need and that they will tolerate. For rapid digitalization it is best to give 1.2 mg. of digitoxin in one dose. Digitalis leaf should be given in divided doses to avoid nausea induced by direct irritation of the stomach. The maintenance dose, too, varies with the individual. When given over long periods of time, 0.2 mg. of digitoxin or 1 cat unit daily is often sufficient. In the presence of auricular fibrillation, the dosage is easily regulated. The ventricular rate is used as a guide, and enough drug is given to maintain the ventricular rate between 70 and 80 a minute. When the rhythm is regular, the heart rate is no guide to dosage. Old people, as a rule, respond to smaller dosages of digitalis than do the young and are more apt to develop toxic poisoning. The appearance of extrasystoles, particularly of coupled beats, is definite evidence of digitalis intoxication. With the first appearance of such signs, the drug should be discontinued until they have subsided. With proper control, vomiting should rarely appear.

When bed rest, limitation of fluids and digitalis do not bring rapid relief, recourse should be had to diuretics. Caffeine and theobromine have a rather feeble diuretic action.

and are rarely used. Aminophylline is more powerful and may be given in doses of 1½ to 3 gr three times a day for a few days. When its use is continued too long it loses its effect. Better than aminophylline is ammonium chloride, given in the form of the 7½ gr enteric coated tablet. Three to four such tablets should be given three times a day. If a still more powerful diuretic is needed, 1 or 2 cc of one of the mercurial diuretics may be given intravenously or intramuscularly. If kidney function is good the dose may be repeated every three to five days, depending on the degree of water retention. The administration of ammonium chloride for 24 hours preceding the injection enhances the action of the mercurial diuretic.

Paracentesis of the chest or abdomen is rarely indicated since mercurial diuretics have come into use. However, when the aforementioned measures do not relieve pleural effusion or ascites, the fluid should be withdrawn, particularly when it is causing distress by its mechanical presence.

After recovery from gross heart failure the patient should be allowed to resume his activities very slowly and guardedly. Incautious haste may result in recurrence of heart failure. After a major episode of heart failure, a four week rest in bed should be prescribed even when the symptoms subside promptly. Return to full activity should be guided by the patient's reaction to physical exertion.

### CARDIAC PSYCHOSES

Serious psychoses occurring as complications of heart disease are not common. It is assumed that they are caused by inadequate nutrition of the brain resulting from the disordered circulation. They appear particularly in older persons with latent or frank cerebral arteriosclerosis. In such persons heart failure may upset the balance of the cerebral circulation.

to such an extent that serious cerebral anoxemia follows. Many patients with chronic cardiac disease eat so poorly for long stretches of time that actual dietary deficiencies may occur. In such patients pellagrous mental changes may develop. Rarely digitalis may induce a psychosis.

The psychic disturbance may manifest itself in several ways and during various stages of the cardiac disorder. Most commonly it appears a few days prior to death, but it may occur many weeks before. It is usually a sign of grave prognostic import, although certain patients recover. The usual picture is that of a grave depressive psychosis. Commonly the patient is convinced that the doctors and nurses wish to do him harm, often he fears poisoning and refuses all food and medication. As a result of his delusion he may become greatly excited in an endeavor to escape from his persecutors, or he may attempt suicide. Accompanying the delusions there may be visual and auditory hallucinations. The motor excitement and restlessness often place a great and deleterious strain on the damaged heart.

Usually the psychic disorder develops during grave myocardial insufficiency and persists with occasional lucid intervals until death. In some instances, however, with the recovery of cardiac function the psychosis disappears. It may develop during convalescence from an attack of heart failure and under such circumstances has been attributed by some authors to the resorption of the edema fluid.

In a masterly manner Head<sup>1</sup> has described the lesser mental changes that often accompany disease of the lungs or heart. Loss of memory and attention is common. Characteristic are hallucinations of vision which usually take the shape of an indistinct draped human form at the foot of the bed.

<sup>1</sup>Head, H. "Certain Mental Changes That Accompany Visceral Disease." *Brain* 24:345, 1901.

and are white, black or gray, never in color. This distinguishes them from hallucinations of the insane which are usually colored. Hallucinations of hearing never include articulate voices but assume the form of tapping, knocking or of bells. Hallucinations of smell are usually accompanied by nausea. In addition patients may experience paroxysms of depression which come on inexplicably. Many are haunted by a vague fear of impending misfortune. In time this mood is followed by a feeling of suspicion that they are being neglected or harmed by their attendants and former friends. Head found that in every case the only constant factor that accompanied these mental disturbances was referred visceral pain. Other factors too play a role for women are affected three times as frequently as men. The explanation of these phenomena can be given best in Head's own words:

Under normal circumstances visceral life takes place outside consciousness. Reflected pain of visceral origin brings in its train all those images and dispositions which exist normally at the fringe of or entirely outside the field of consciousness. All those sensations that are associated with visceral activity which do not exist in consciousness under normal conditions come to the surface. The patient's character appears to be altered for the content of his consciousness is changed. He will become moody at one time unduly exalted at another depressed without cause. Reason is displaced and he is the victim of each passing wave of feeling tone and he will have lost control over the expression of his emotions and of his temper.

That barrier which the normal mind sets between conscious life and that of the viscera the integrity of which depends on a high potential of vitality in the nervous system has been broken down.

The treatment of cardiac psychoses is primarily treatment of the accompanying heart failure. Oxygen should always be given to lessen cerebral anoxemia. When there is reason to suspect dietary insufficiencies nicotinic acid should be given,

by mouth or intravenously, 300 to 500 mg a day. If there is no improvement within two or three days the drug should be discontinued. With improvement, the medication should be continued, but after the first week 50 mg three times a day is sufficient.

### THROMBOPHLEBITIS OF THE LEG VEINS AND PULMONARY EMBOLISM

Pulmonary embolism arising from a thrombus in the veins of the legs is a common and hazardous complication in bedridden patients who are over the age of 50. It occurs not only after operations but in all kinds of illnesses, particularly in the obese and in patients with heart failure. Thrombophlebitis of the veins of the lower extremities and of the pelvis becomes increasingly common with advancing years. Barnes<sup>2</sup> reported that at the Mayo Clinic 70 per cent of patients who died of postoperative pulmonary embolism were over 50 years of age and that only one was less than 30. Hunter<sup>3</sup> found thromboses of the calf veins in 53 per cent of 351 cases seen at autopsy. The incidence of pulmonary embolism with venous thrombosis was 6 per cent.

In 100 of the cases with venous thromboses, 5 per cent had fatal and 20 per cent nonfatal pulmonary emboli. Somewhat over 2 per cent of all deaths of patients in hospitals are due to pulmonary embolism.

The chief factor responsible for peripheral venous thrombosis and subsequent pulmonary embolism is bed rest. The more immobile the patient, the more readily does thrombosis

<sup>2</sup> Barnes, A. R. "Pulmonary Embolism." *J. A. M. A.*, 109:1347, Oct. 23, 1937.

<sup>3</sup> Hunter, W. C. et al. "Thromboses of the Deep Veins of the Leg." *Arch. Int. Med.*, 68:1, July, 1941.

occur. Thrombi may form after a few days of confinement. In England not a few cases were encountered in elderly people who were compelled to sleep in cramped positions in air raid shelters. Operation and traumatic injuries are common antecedents of thrombosis and embolism. It is not generally recognized that heart failure is one of the chief causes predisposing to thrombophlebitis and pulmonary embolism. Venous thrombosis and embolism are so common in bed-ridden cardiac patients that they should always be watched for. Thrombophlebitis is more frequent in the winter and in cold climates.

Immobility as well as heart failure lead to slowing of the blood stream in the peripheral veins. The venous blood stream is maintained by the arterial head of pressure, by the contractions of the muscles, by free respiration which creates a negative pressure in the thorax and by the descent of the diaphragm which milks blood out of the abdominal structures. All of these factors are reduced by immobility in bed and the flow of blood is further retarded by shock or by cardiac insufficiency.

Fully 95 per cent of venous thromboses occur in the deep veins of the calf. From here the thrombus extends up to the femoral and external iliac veins. More rarely the pelvic veins or the prostatic venous plexus is involved. When one of the larger, more superficial veins is thrombosed the diagnosis is easy. There is pain, swelling and tenderness along the affected vein, the leg becomes swollen, and the overlying skin may be reddened. The patient develops fever. More often than not these gross evidences of venous thrombosis are lacking, the patient may complain of a little pain in the calf of the leg, and a pulmonary embolus first calls attention to the peripheral venous thrombosis. Only by careful daily observation can an early diagnosis be made. Many cases run

their course with minimal or no pain and no local swelling. The calf or thigh muscles may be tender to pressure, measurement of the circumference of the legs may reveal slight enlargement of one extremity, there may be slight fever and leukocytosis. The sedimentation of the red blood cells is

The films are difficult to interpret and often lead to erroneous conclusions.

Often a pulmonary embolus gives the first warning of the presence of thrombophlebitis. The occurrence of a pulmonary embolus is presumptive evidence of thrombophlebitis of the veins of the legs and calls for careful examination of the legs. Even in the absence of physical signs the presence of thrombophlebitis in the legs should be assumed unless there is another patent source of the embolus.

The symptoms of pulmonary embolism are rarely the classic ones of hemoptysis and pleural friction rub followed by consolidation of the lungs. Even dyspnea is not a constant sign. The chief evidences are sudden shock, faintness, sweating and a rapid pulse. Often weakness and anxiety are the sole symptoms. There may be chest pain, usually in the axilla. Hemoptysis is relatively infrequent, and lung signs may or may not develop. Roentgenography of the chest should be freely used as an aid in diagnosis. A rise in temperature is the rule. The first embolus is usually a small one. One or more of such nonfatal embolic episodes usually precede a lethal attack. Pulmonary embolism occurs in about one of three patients with thrombophlebitis, and in one of 25 cases the attack is fatal.

In certain cases the textbook signs of dyspnea, pulmonary consolidation and hemoptysis develop. Bronchopneumonia

is often erroneously diagnosed. A massive embolus may cause instantaneous death. A large nonfatal embolus gives rise to the signs and symptoms of acute cor pulmonale, characterized by acute dilatation and strain of the right ventricle. In such cases there is marked dyspnea, often with cyanosis. Increased cardiac pulsation may be visible in the second and third interspaces to the left of the sternum in the region of the conus of the right ventricle. A systolic murmur is audible in this area and the pulmonic second sound becomes accentuated. The electrocardiogram may show a characteristic pattern with an S wave in lead 1, the ST segment depressed, the T wave diphasic in lead 2 and the RT segment elevated and the T wave negative in lead 3. There is usually a Q wave in lead 3. The electrocardiogram may be confused with that of an infarct of the posterior wall of the left ventricle. If the patient recovers, the electrocardiogram returns to its original pattern.

*Treatment*—Prevention of thrombophlebitis of the veins of the legs is possible in a large proportion of cases. Measures should be directed to preventing slowing of the venous return flow from the legs. Elderly persons should not be confined to bed more than is absolutely necessary, when in bed they should be encouraged to move about freely, and if they are disabled their position should be changed frequently. They should be compelled to flex and extend their feet, legs and thighs several times a day, and if this is impossible they should be given centripetal massage of the legs twice daily. Frequent deep breathing exercises should be encouraged. Heart failure should be actively combated.

Thrombophlebitis is so common in older debilitated patients that some experienced surgeons recommend prophylactic bilateral femoral ligation before operation for all patients over 65. There is much to be said for this procedure.



If thrombophlebitis of the leg veins develops despite prophylactic treatment, and more particularly if a pulmonary embolus occurs, even in the absence of physical signs of thrombophlebitis, immediate active intervention is indicated.<sup>4</sup> If the site of the thrombosis is known, the superficial femoral vein of the corresponding leg should be exposed and opened, the thrombus removed by suction and the vein divided between ligatures. Often it is necessary to do a bilateral femoral vein ligation because it is impossible to determine which veins are involved. Indeed, this is probably the wisest routine procedure in all cases, for the thrombosis is so commonly bilateral.

If the site of the thrombosis cannot be determined or if for some other reason operation is not feasible, consideration should be given to the use of anticoagulants to arrest upward extension of the thrombus. The best preparation is heparin. It has the disadvantages that it is very expensive, must be given intravenously and requires constant check on the coagulation time of the blood. It may be given in intermittent doses of 100 mg. three times a day, or approximately the same quantity may be given by intravenous drip throughout the 24 hours. The dosage should be adjusted to maintain the clotting time of the blood between 30 and 45 minutes. Heparin prevents extension of the thrombus, but it is difficult to know how long to continue treatment. A week is probably the minimum time. In several of my cases there was renewed extension of the phlebitis after the administration of heparin had been stopped. Again resumption of treatment promptly arrested the venous thrombosis. Dicoumarin, an anticoagulant that can be given by mouth and is inexpensive, is still in the experimental stage. Its administration must be carefully

<sup>4</sup>Allen, A. W. et al. "Venous Thrombosis and Pulmonary Embolism," J. A. M. A., 128:397, June 9, 1945.

controlled by determinations of the prothrombin in the blood

When there is massive pulmonary embolism  $\frac{1}{2}$  gr of papaverine hydrochloride should be given intravenously at once and repeated if necessary. This helps relieve secondary spasm in the pulmonary artery. Morphine should be administered to allay pain and oxygen to ease the respiration and relieve anoxemia. Subsequently full doses of digitalis should be given to combat heart failure. In all cases of pulmonary embolism sulfadiazine or penicillin should be given at once because bronchopneumonia is an inevitable complication.

## Chapter Ten

# DISEASES OF THE LUNGS

### PULMONARY EMPHYSEMA

TWO TYPES of pulmonary emphysema are distinguished senile or nonobstructive emphysema, and essential or large lunged emphysema

*Senile emphysema* is encountered in older individuals and has been interpreted as reflecting a degenerative process of the lungs. When the thorax is opened at autopsy the lungs collapse. They are small, friable and anthracotic. Microscopic examination reveals broken down and enlarged pulmonary alveoli, with atrophy of the septa and no increase in the elastic tissue of the lung. The chest is not large, not barrel-shaped. The spine is straight, and there is a degenerative change in the intervertebral disks. This elevates the ribs and increases the size of the thoracic cage, and alters the position of the lungs. Kountz and Alexander<sup>1</sup> believe that the skeletal change is the primary one. Calcification and ossification of the costal cartilages and ankylosis of the costovertebral joints are often marked. The diaphragm is not depressed, and the diaphragmatic excursions may be extensive. Indeed, in senile emphysema, in contrast to the essential form, the pa-

<sup>1</sup> Kountz, W. B., and Alexander, H. I. "Nonobstructive Emphysema" J. A. M. A., 100:551, Feb. 25, 1933

tient's breathing is largely abdominal, effected by the action of the diaphragm rather than by the intercostal muscles. Interference with respiratory function is less than in essential emphysema. There is no past history of lesions inducing bronchial obstruction. However, the several processes lead to the same physiological end result as that which takes place in essential emphysema—a progressive impairment of the respiratory function and the vital capacity.

*Essential emphysema* commonly first manifests itself in younger persons, often at the age of 40. The lungs are large and voluminous, pushing the diaphragm down, elevating the ribs and covering the anterior aspect of the heart. Large bullae, representing huge air sacs, are often seen, particularly along the anterior lung margins. Microscopic examination reveals broken down and enlarged alveoli with much secondary increase of elastic tissue fibers. The bronchi are usually inflamed and injected and may be dilated. The chest is deep and wide and the spine is curved into a kyphosis, so that the chest assumes the classic barrel shape. The neck is short, the scapulae ride high. Calcification of the costal cartilages progresses with the years. Because of the large volume of the lungs, displacing the diaphragm downward, the diaphragm moves very little with respiration. Respiration is effected largely through contraction of the intercostal muscles and the accessory muscles of respiration, and the costal margins move inward with each breath. Because of the structural alterations of the lung tissue, which make it less efficient as a medium for the exchange of gases between the alveoli and the blood, and because of the loss of elasticity of the lungs and the impaired mechanism of the respiratory musculature, there results marked impairment of respiratory function. The two chief manifestations of this are dyspnea and cyanosis. The chronic anoxemia resulting from the impaired respiratory

function leads to secondary polycythemia. The blood volume is not increased, and the hemoglobin and red cell contents do not reach the levels encountered in primary polycythemia. The hemoglobin content rarely rises above 110 per cent, and the red cells do not exceed 6,500,000 per cu. mm. The obliteration of much of the capillary bed in the lungs, due to atrophy and destruction of the alveolar septa, increases the peripheral resistance in the pulmonary blood circuit and increases the blood pressure in the pulmonary arterial tree. This leads to atherosclerosis in the pulmonary arteries, in both the large and the small vessels. The work of the right ventricle is augmented, so that in the course of time hypertrophy and dilatation of this chamber of the heart occur. Eventually right heart failure, with tricuspid insufficiency and dilatation of the right auricle may ensue.

In both senile and essential emphysema hereditary factors are very evident, so that one encounters multiple cases in families. The development of essential emphysema is favored by the occurrence of pulmonary infections and by lesions which cause partial obstruction of smaller bronchi. Thus emphysema is an invariable sequel of persistent bronchial asthma and commonly follows recurrent attacks of bronchitis.

*The symptoms of senile and essential emphysema are identical, varying only in degree, so they will be discussed together.* Senile emphysema is much slower in its evolution and for years leads to little impairment of respiratory function.

The appearance of the patient with fully developed emphysema is so typical that it makes the diagnosis self-evident. One sees a middle-aged man with high color, often cyanotic, puffing on slight exertion and subject to frequent cough. The chest is deep and broad, the neck short, the back curved and the shoulders stooped forward. The neck veins are full. The finger-nails show increased incurvation, and often there is

true clubbing of the fingers. There is little or no abdominal component of the respiratory movement, the epigastrium does not move out with each inspiration. The chest moves up and down as though in one piece.

Percussion yields a hyperresonant, tympanic note all over the lung fields. The areas of cardiac flatness and dullness are obliterated by the interposition of the voluminous lung between the heart and the anterior chest wall. The diaphragm is pushed down so that pulmonary resonance extends below the tenth rib posteriorly. The general hyperresonance obscures lesions that in a normal chest would give rise to dullness or flatness. Thus infiltration or fibrosis of the lung, or even a pleural effusion of moderate size, is easily missed in a markedly emphysematous chest.

Auscultation yields the characteristic sign of pulmonary emphysema, namely, prolonged expiration. By watching the patient one sees that inspiration is quick and short, expiration lengthy. The respiratory note is low-pitched and soft and in advanced cases may be almost inaudible. Commonly, wheezing and sibilant râles are scattered throughout the chest, and at the bases posteriorly sticky moist râles are often heard. Complicating bronchitis, the condition that so often sends the patient to the doctor, accentuates all of these signs. Expiration becomes still more prolonged, the breath sounds are fainter, and râles of all sorts are found widespread in both lungs from apex to base. At times they may be restricted to one lower lobe.

The roentgen picture, too, is typical. The lung fields are clear and brightly illuminated, they do not become darker on expiration, and the shadow of the heart and great vessels stands out clearly in contrast. The clavicles are high, and intercostal spaces wide and horizontal. The dome of the diaphragm is flattened, and its excursions greatly diminished.

Physical signs referable to the heart are difficult to elicit. The size of the heart cannot be mapped out because of the general hyperresonance the heart sounds are dulled and distant because of the cushion of lung interposed between it and the chest wall. In normal chests that are not too thick walled the quality of the heart sounds gives a significant indication as to the state of the heart muscle. This is not so in the presence of emphysema. In such patients an accurate cardiac diagnosis cannot be made without use of the fluoroscope and the electrocardiograph. Such examinations are all the more important because of the frequent association of arteriosclerotic heart disease and pulmonary emphysema.

The chief symptoms are dyspnea and cough. At first the cough is noted chiefly in the morning on arising and is relieved when the accumulated nocturnal secretions have been expelled. Gradually through the years the cough becomes more persistent and continuous and the dyspnea more disabling. In advanced cases it may be completely incapacitating. Patients with pulmonary emphysema are peculiarly subject to attacks of bronchitis and usually contract several such infections every winter. Each attack leads to added damage to the lungs. The infection itself favors further weakening of the pulmonary parenchyma and the swelling of the bronchial mucosa brings about increasing expiratory obstruction which induces greater dilatation and breaking down of the wheals of the lung. Furthermore during such a bronchitic seizure the blood pressure in the pulmonary arterial tree is raised and the strain on the right ventricle is increased so that commonly heart failure occurs during such an episode. Bronchopneumonia is a frequent sequel of such a bronchial infection and gives rise to fresh damage of both lung and heart and often causes death.

Pneumothorax may occur when a large emphysematous

bleb ruptures, usually during a coughing spell or following sudden physical exertion. The symptoms and physical signs do not differ from those of pneumothorax encountered in younger individuals. Usually with immobilization in bed the air in the pleural space is absorbed in two to four weeks and no permanent evil results follow. Certain patients repeatedly have such attacks. In other cases a valve-like action of the ruptured lung is established, so that with each inspiration air enters the pleural space and cannot escape into the lungs with expiration. So a tension pneumothorax is set up, an increasing volume of air is pumped into the pleura, the mediastinum is displaced to the healthy side, and severe even dangerous respiratory distress may ensue. In such cases it may be necessary to withdraw some air from the affected pleural space. This is done by attaching a rubber tube to a chest aspiration needle. The free end of the rubber tube is inserted into a jar of water, and the needle is inserted into the pleural space. Thus the escaping air is allowed to bubble up through the water. In cases in which the air is not absorbed or in which it reaccumulates, it may be necessary to attempt to seal the hole in the lung. This can sometimes be done by injecting 20 cc of 20 per cent glucose solution into the pleura that is distended with air. The resulting reactive inflammation may close off the tear in the lung. Such a procedure, of course, must be done with great care under aseptic precautions, and the patient must be immobilized in bed, lying on the affected side for several weeks.

In certain cases of pulmonary emphysema, individual emphysematous blebs may reach a huge size, forming what are known as *air cysts* in the lung. Such a cyst may displace a whole lobe of a lung. It can be diagnosed only by the roentgen picture and is often mistaken for pneumothorax. Air cysts give rise to no symptoms and are susceptible to no treat-





FIG. 18 -Pneumothorax complicating pulmonary emphysema. Note clear area delineated by arrows.

ment Their recognition is important only from the point of view of differential diagnosis They may persist unchanged and roentgen pictures taken at long intervals may be identical

The heart Symptoms arising in the heart in patients with pulmonary emphysema are caused most frequently by associated coronary artery sclerosis or hypertension These symptoms do not differ from those in patients with uncomplicated heart disease except that the right ventricular strain caused by the pulmonary disease is more apt to lead to right heart failure In long standing cases of emphysema particularly when there is much fibrosis of the lungs and when there have been repeated bouts of pulmonary infection right heart failure eventually occurs In its earlier stages it is difficult to diagnose for both dyspnea and fullness of the neck veins may be due to the emphysema alone It is in such cases that measurement of the circulation time of the blood is of particular value If the circulation time from the arm to the tongue is normal that is less than 18 seconds then it may be inferred that the symptoms are due to the pulmonary lesion But if the circulation time is prolonged it is evident that myocardial failure has set in and is contributing to the symptomatology

When right heart failure occurs the heart dilates rather rapidly dyspnea and cyanosis become intense the neck veins and the liver become engorged and there may be edema of the legs The lungs may eventually become congested and pleural transudate may accumulate The prolonged and intense stasis in the peripheral circulation increases the work of the left ventricle until it too fails Figure 19 shows the chest x ray of a man aged 45 with advanced pulmonary emphysema whose heart was compensated The heart is not enlarged but the arch of the pulmonary artery on the left bor-

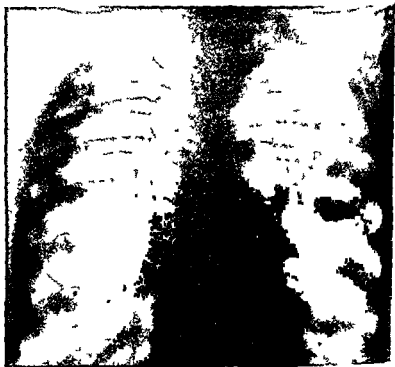


FIG 19 -Pulmonary emphysema with hypertrophy of the conus arteriosus and of the right ventricle of the heart

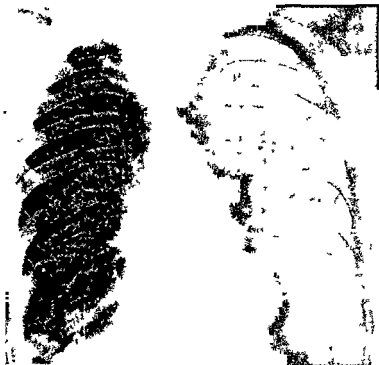


FIG 20—Same patient as in the preceding six months later Great cardiac dilatation accompanying heart failure

der of the heart is prominent, suggesting dilatation of the pulmonary artery and hypertrophy of the right ventricle. Figure 20 was taken exactly six months later, when the patient had suddenly become gravely ill with severe heart failure that led to death within a few weeks. Generalized cardiac enlargement is apparent. Such a degree of cardiac dilatation rarely recedes. Treatment of severe right heart failure in patients with pulmonary emphysema is exceedingly unsatisfactory. Cardiac compensation usually cannot be restored, and death follows within a few weeks or months.

**Hernias.** Hernias commonly develop in patients with emphysema and chronic bronchitis. The cough, by forcibly raising intra abdominal pressure, gradually forces intra-abdominal structures through points of weakness in the abdominal wall. In older patients the giving way of the abdominal wall is favored by the increasing weakness and atrophy of the body musculature. Usually one encounters inguinal and umbilical hernias. In patients with operative scars ventral hernias commonly develop. Operative treatment is uniformly unsuccessful, for the persistent cough speedily leads to a recurrence of the hernia. An appropriate truss should be provided.

**Treatment.**—No treatment can restore the emphysematous lung to normal, but careful supervision and regulation of the patient's life can arrest the progress of the disease, prolong life and postpone complications. The two elements that favor progression of the lesion are pulmonary infection and factors such as cough that tend to increase intrapulmonic pressure. Cough is favored by bronchial infection, by exposure to dust and by smoking. These patients must avoid dusty occupa-

chial asthma is a direct cause of many cases of pulmonary emphysema so that it is essential to try to prevent asthmatic seizures. In older patients one rarely finds a specific allergen that is responsible for the asthmatic attacks. There may be multiple sensitivities but in the majority of the older patients with well established involvement and emphysematous changes in the lungs we are dealing with an infectious asthma which is difficult to eradicate. Treatment of sinus infection and removal of polyps rarely lead to permanent relief. At times autogenous vaccines prepared from the patient's sputum are helpful.

Prevention of recurrent attacks of bronchitis is more easily talked about than accomplished. Of course the patient should avoid exposure to raw cold inclement weather and drafts. When he is obliged to go out in rainy or cold weather he should protect himself against the elements. He should avoid other persons who have colds or grip for bronchitis commonly follows an apparently mild coryza.

The best measure one which unfortunately cannot be carried out by the majority of patients is removal to a warm equable climate. Many of these patients do well in Florida or in southern California. Others do better in the drier climate of New Mexico or Arizona. The emphysematous patient does not tolerate the cold raw northern winters.

Many patients with pulmonary emphysema obtain symptomatic relief by wearing an appropriate abdominal belt.<sup>2</sup> It will be recalled that in these patients the diaphragm is pushed down in the position of inspiration and that its respiratory movements are small. The diaphragm can be pushed up toward its expiratory position by a properly adjusted abdominal belt. From this position contraction again occurs.

<sup>2</sup> Alexander H. L. and Kountz W. B. Symptomatic Relief of Emphysema by an Abdominal Belt. *Am J M Sc* 18: 697 May 1934.

The patient experiences increased ease of respiration, and the vital capacity of the lungs may increase by as much as 40 per cent

Regulation of the bowels is of particular importance in these patients, because constipation makes them strain at stool, and this in turn raises the intrapulmonic pressure and so favors progression of the emphysematous process

Breathing exercises often bring about symptomatic improvement, increase vital capacity and lessen dyspnea. The exercises are directed to teaching the patient diaphragmatic breathing. They are described in greater detail in the section on asthma (p 252)

Medication is not curative in pulmonary emphysema. It is designed to give symptomatic relief from the cough. When there is much wheezing, due to bronchial spasm or congestion, ephedrine hydrochloride  $\frac{3}{4}$  gr combined with phenobarbital  $\frac{1}{2}$  gr is often helpful. This may be given from two to four times a day. In the presence of hypertension or coronary disease it should be administered cautiously. In my experience, expectorants are of little value, and too often they upset the stomach. Small doses of potassium iodide, 2 to 5 gr three times a day, may be given. When the cough is at all severe, codeine is the only drug that will help. The dose should be the smallest that will alleviate the cough. It is best to try doses of  $\frac{1}{4}$  gr first and to give larger doses only if necessary. Nothing will stop the characteristic morning cough by which the patient clears the lungs of the secretions accumulated during the night. Their expulsion, after

the sputum is expectorated, liquefies and inspissates the mucous membranes and inspissating the secretions

Any slight cold or upper respiratory infection should be treated as a serious complication, for in emphysematous pa-

tients bronchitis or bronchopneumonia commonly follows. The patient should be put to bed in a room of equable temperature and free from drafts. The diet should be light, the bowels should be kept open. If bronchitis occurs, treatment with penicillin or sulfadiazine should be instituted at once. It is impossible to distinguish between severe bronchitis and early bronchopneumonia in these patients, and it is dangerous to await the development of frank signs of pneumonia. Often a serious infection can thus be aborted. If breathing is difficult, steam inhalations may be grateful. A sedative cough mixture, such as Stokes' expectorant, should be given. If necessary, codeine may be added to it. If there is much respiratory difficulty, and especially if there is increasing cyanosis, oxygen may give great relief. This may be administered by nasal catheter or through a B L B mask. Morphine should be avoided because it depresses respiration and favors accumulation of secretions in the lungs and development of bronchopneumonia. With the onset of bronchitis, digitalis should be given routinely. The bronchitis places an additional strain on the right ventricle and may induce heart failure. Digitalis may prevent this. Further, by improving the circulation through the lung it hastens resorption of the inflammatory process. It is best administered as the  $1\frac{1}{2}$  gr., 1 cat unit tablet. For one week, two tablets a day should be given. After the first week the dose should be reduced to one tablet a day. In the presence of fever and dyspnea the patient should be kept quiet, usually in bed, but his position should frequently be changed and he should be encouraged to sit up in bed. Often he is more comfortable in an easy chair. As soon as possible, when the temperature has subsided, or when it does not exceed 100 F., he should be up part of the day. This promotes deeper respiratory excursions and helps to clear the lungs. Older patients are greatly prostrated following an at-



tack of bronchitis and it may take weeks for them to recover their strength. The effect of so called tonics is notoriously difficult to estimate, but many patients are apparently helped during convalescence by taking 2 mg of thiamine hydrochloride and 50 mg of niacin three times a day. At times strychnine sulfate 1/30 gr seems to improve their strength.

Aerosol administration of penicillin may clear up bronchial infections. A nebulizer like the DeVilbiss No. 40, filled with 200,000 units of penicillin in 2 cc of water is connected with a small oxygen tank equipped with a reducing valve. There is a hole in the tubing proximal to the nebulizer. The oxygen is turned on and the opening of the nebulizer placed in the mouth. Inhaling the patient closes the hole with his finger, exhaling he opens it, and continues until the penicillin solution is exhausted. This is repeated several times daily.

If bronchopneumonia develops it is treated as any other bronchopneumonia (see p. 389).

Severe *heart failure* complicating pulmonary emphysema is very grave. The patient should be put at complete rest in bed. His fluid intake should be limited to 1,000 cc in 24 hours. If the neck veins are full and the liver is engorged and if the red blood cell count is high, one or more phlebotomies of 500 cc give great relief. Oxygen should be administered continuously until the heart failure is controlled. Digitalis 1½ gr twice a day, should be administered and continued at this dosage for at least two or three weeks. If the patient has had no previous digitalis medication, larger doses may be given the first day. One may give an initial dose of 6 gr and a second dose of 6 gr six hours later. If there is insufficient response to these measures a mercurial diuretic 2 cc of mercuripurin, should be given intravenously or intramuscularly. This may be repeated as indicated, every three to seven days. Severe heart failure in patients with emphysema has a

poor prognosis. Rarely is compensation fully restored. Death commonly follows in weeks or months.

*Operation* in an emphysematous patient is hazardous because of the danger of postoperative pneumonia. Ether is best avoided as an anesthetic because of its irritation of the lungs. Local anesthesia, avertin, gas oxygen, cyclopropane and ethylene are the anesthetics of choice. Use of plaster spicas that limit the movement of the chest or diaphragm is dangerous and should be avoided. Such immobilization of the chest will almost certainly induce a bronchial infection.

#### PULMONARY FIBROSIS AND BRONCHIECTASIS

Pulmonary fibrosis is common in older individuals and is usually associated with emphysema. It results from repeated pulmonary infections and is localized chiefly in the lower lobes. When such fibrosis becomes more extensive, bronchiectasis is found with it. The typical bronchiectatic dilatations in older persons are usually not as great or as destructive as those found in younger individuals following suppurative or interstitial pneumonia. In older persons repeated attacks of bronchitis and bronchopneumonia lead to peribronchial inflammation and fibrosis and weakening and dilatation of the bronchial wall.

The disturbance of respiratory function and the symptoms resemble those of advanced pulmonary emphysema. However, cough productive of much sputum is one of the chief complaints. The sputum is rarely foul and is not as copious as in younger persons with bronchiectasis. The cough is most aggravated in the morning on arising and persists until the bronchi have been cleared of the nocturnal accumulation of secretions. Incurvation of the finger nails or clubbing of the fingers is common. These patients are particularly subject to

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occurs after the age of 50 and is usually unilateral. Previous trauma or fracture may favor its development. The head of the femur is deformed and there is bone production about the acetabulum so that the head of the femur no longer fits its socket. There is a slow onset of pain in the hip joint with stiffness and resulting limp and limitation of abduction and external rotation. Commonly, obturator or sciatic neuralgia results so that the pain is referred to the groin or to the knee. Roentgen examination may reveal a narrowed joint cavity, exostoses or flattening of the head of the femur.

Heberden's nodes are bony exostoses that appear on the proximal ends of the terminal phalanges of the fingers as a result of local periostitis. They may develop rather rapidly with local pain and redness of the overlying skin. Often light trauma to the affected areas arising from ordinary use of the hands will bring on swelling, redness and pain that last a number of days. In other cases Heberden's nodes may come on insidiously with little pain. The patient complains chiefly of the deformity. Stecher found Heberden's nodes in almost one quarter of persons between the ages of 50 and 69 and in over one third of those over 70. Heberden's nodes occur chiefly in women. There is a distinct familial predisposition to this disorder, but usually only sisters are affected while brothers are free from the disease. Inheritance appears to take place only through the mother.

Malocclusion of the teeth from faulty natural or artificial dentures causes displacement of the condyle of the mandible and resultant lesions of the temporomandibular joint often with irritation of the auriculotemporal nerve. Facial neuralgia, tinnitus and headache may result and these can be relieved by appropriate dental reconstruction.

*Treatment.*—This is difficult and taxes the patience of both physician and patient. If it is intelligently planned and

carried out with persistence, good results can be achieved. The first indication is to spare the joints that are being damaged by excessive use and by malfunction brought on by static disorders of the body. Complete bed rest is rarely essential, but the patient should safeguard and rest his joints as much as possible. He should not walk too much and, if the knees are involved, should avoid climbing stairs. If there is spondylitis, he should avoid bending and lifting. If there are Heberden's nodes, activities that traumatize the fingers, such as heavy housework or manual labor, should be eschewed. Blows to the fingers by striking the hand carelessly against a hard object often cause an acute inflammatory reaction, with subsequent increased growth of the node.

Obese patients should be encouraged to lose weight. The joints should be protected by padding and splints. In advanced cases, a corset, a painful spine may need a brace. Often the orthopedist must be called on for help. At times desiccated thyroid in doses of  $\frac{1}{2}$  to 1 gr. a day seems to be of assistance. The various modalities of physical therapy, particularly the several forms of heat and massage, are very useful. Medication has no effect on the course of the disease but is often needed to relieve pain. For this aspirin, phenacetin and codeine are the most useful drugs.

### RHEUMATOID ARTHRITIS

Rheumatoid arthritis is the preferred term for the condition also known as infectious or atrophic arthritis. It is essentially a disease of the younger years of life. Fully 80 per cent of cases have their onset between the ages of 20 and 50. The peak of cases occurs at about age 35. The disease runs a prolonged course, and many patients survive into the sixth decade or longer. Individual cases may have their onset in the

later years of life I have seen a typical case that made its first appearance in the patient's eighty-fifth year. Rheumatoid arthritis is three times as common in women as in men. Although it is probably an infectious disease, the causal organism has not been identified. For many years it was believed that foci of infection in the teeth, tonsils, prostate or gallbladder were the cause. This view has been relinquished even by its former most ardent advocates, after hundreds of patients have been operated on and mutilated without benefit.

The initial pathological lesion is an inflammation of the synovial membrane, which becomes thickened by exudative and proliferative processes. Swelling and inflammation extend to the periarticular tissues. The proliferating synovial tissue grows over the articular cartilage, invades and gradually destroys it. When opposing surfaces of joint cartilage are covered by this pannus of inflamed granulation tissue, adhesions form. The end stage is marked by fibrous or bony ankylosis and subluxation of the joints.

The onset may be sudden, with acute swelling, redness and painfulness of several joints and high fever. There is usually symmetrical joint involvement. More often the onset is not so stormy but is marked rather by an insidious indolent inflammation of many joints in slow succession. Any joint may be involved, but the joints of the fingers, the wrists and the knees are the favorite localizations. Muscle atrophy in the regions around the joints and trophic disorders of the skin of the extremities quickly develop. Thickening of the periarticular tissues with atrophy of the muscles above and below the joint gives the part its typical spindle shaped appearance. The skin becomes shiny and smooth, red or dusky purplish, and there may be trophic disturbances of the nails. Systemic manifestations are fever, loss of weight, secondary anemia and a rapid sedimentation rate of the red blood cells.

The lymph nodes draining the affected joints are often enlarged. Subcutaneous nodules are found in one fifth of the cases. They occur over bony prominences—the olecranon, the sacro iliac joints, the spinous processes—and may persist for years. Valvular heart disease, indistinguishable from rheumatic heart disease, is a frequent complication, especially at younger ages.

Röntgen examination at first reveals no changes in the contours of the joint surfaces, only swelling of the soft tissues and decalcification of the bones. In the later stages of the disease, as a result of destruction of the articular cartilage, the joint spaces become narrowed, and punched out areas may appear in the bones near the joint surfaces. In the final stage there are bone production and ankylosis.

Unlike rheumatic fever, the disease does not have a self limited course with restoration of the joints to normal. The arthritic manifestations last for weeks or months, vary in intensity, shift from one joint to another and usually leave some permanent joint deformity. Eventually contractures and subluxations result. The constitutional symptoms persist, too. The patient loses weight progressively and may become quite emaciated and anemic. The sedimentation rate of the red blood cells is one of the best indicators of continued activity of the infectious process. Until it reaches normal figures the disease is not arrested. Even when the disease is completely quiescent one cannot speak of cure, for recurrences are common. There may be free intervals of months or years, but as a rule sooner or later the arthritis again lights up.

*Treatment*—During the active stage of the arthritis rest in bed is essential. The bed should be firm and the mattress should not sag. Every effort should be made to maintain the nutrition of the patient and to combat the wasting that commonly occurs. The diet should have a high caloric value and



should contain a full complement of the accessory foodstuffs. If the patient is anemic, repeated blood transfusions are indicated. Iron is not helpful until the infectious stage has passed. Joints that are acutely inflamed should be immobilized in light splints, but these splints should be removed daily and the joints should be subjected to gentle passive motion to prevent ankylosis. Codeine or salicylates are given to relieve pain, but have no effect on the course of the disease. As the acute stage subsides, various forms of physiotherapy may be employed. Heat often gives great relief. An infra red lamp may be used, 30 minutes once a day, or an electric pad or a baking apparatus. If the patient can be moved without too much pain, hot baths for 20 to 30 minutes once a day at temperatures between 95 and 102 F. often give great relief. Diathermy or short wave treatments are sometimes helpful. At times heat in any form increases the pain in the joint. In such cases it should not be used until the lesion has become more quiescent. Gentle massage and passive movements of the joints should be carried out immediately after heat treatments.

Every effort should be made to prevent contractures and deformities. If ankylosis develops, the joint should be immobilized in its optimum functional position. The knee joint, for instance, should be fully extended, not flexed, the elbow, on the other hand, should be flexed at about 90 degrees. When the disease has become quiescent, certain deformities may be corrected by operation.

Nonspecific protein therapy is helpful in some cases. This may be given in the form of boiled skim milk, 5 cc intramuscularly every day or every other day. Intravenous injection of typhoid vaccine is more effective. Such injections often give rise to chills or more severe reactions and should not be employed in patients who are too old or who have

arteriosclerosis of the coronary, cerebral or peripheral arteries. The initial dose of typhoid vaccine is usually 10 000 000 organisms. The dose is increased by about 50 000 000 with each successive injection. The succeeding injection is given 24 hours after the febrile reaction to the previous injection has worn off.

Gold seems to be establishing itself as a valuable therapeutic agent in rheumatoid arthritis. Its use is still in the experimental stage and serious toxic reactions occur in 20 per cent of cases. The reader who wishes to employ it is advised to consult the most recent authoritative literature.

Removal of foci of infection is no longer the center of all therapeutic endeavor in cases of rheumatoid arthritis. In rare instances a recently inflamed solitary joint will clear up after extraction of an infected tooth, but as a rule removal of foci of infection has no effect on the course of the disease. I have seen a serious exacerbation of symptoms following tonsillectomy or wholesale tooth extraction. Infected teeth or other structures in the body that give rise to local symptoms should be treated or removed with a view to relieving the local condition, not with the hope of curing a systemic rheumatic infection. Thus, in a patient with repeated attacks of tonsillitis and enlarged tonsillar lymph nodes, tonsillectomy is indicated whether or not he has rheumatoid arthritis.

No direct effect of climate on rheumatoid arthritis has been demonstrated, but the disease is less frequent in tropical and semitropical regions. Certainly these patients feel better if they can escape the northern winters for a sojourn in the south.

### FIBROSITIS

Fibrositis is a term applied to an ill defined syndrome embracing a variety of conditions characterized by aches and

pains in muscle and fibrous tissues. The laity speaks of muscular rheumatism. Pain, stiffness and soreness appear suddenly in a muscle, around a joint or in a fascial sheath. There is no fever or constitutional reaction. The pain is worse in the morning after a night's immobility and gradually lessens with use of the affected structures. The physical findings consist of tenderness over the affected area, at times some muscle spasm and occasionally fibrous nodules. These nodules occur most often over the sacro iliac joints or in the occipital region at the base of the skull. In the latter region these nodules may cause severe headache, sometimes called nodular headache, caused by involvement of the great occipital nerve. Lumbago is fibrositis of the lower part of the back.

The symptoms may be exceedingly acute, causing complete disability for a few days, or they may be milder but persistent. The patient complains of a dull ache, aggravated by movement. The parts involved are stiff, and there may be muscle spasm. The pain commonly shifts from one part of the body to another. Exposure to cold and wet and to drafts often precipitates acute episodes of pain. These patients are very sensitive to changes in weather, and on dull, rainy days their pains are apt to be worse.

The cause of fibrositis is unknown. Rarely, infected sinuses or other foci seem to be responsible for the symptoms. Although the condition causes much annoyance and occasional acute suffering, it is not a progressive disease. Symptoms often recur, but permanent disability does not result.

*Treatment*—This is concerned primarily with general hygienic measures to keep the patient in good general condition. Exposure to wet or to sudden changes of temperature should be avoided. The diet should be adequate, the bowels regulated. The patient should avoid fatigue and obtain enough sleep. In the acute stage, rest is the best measure.

The affected part should be warmly wrapped to protect it against chilling and drafts. Aspirin, phenacetin or codeine given in suitable doses controls pain. Counterirritation with plasters, oil of wintergreen or surface heat lessens the pain. Within a few days, when the acute symptoms lessen, heat and massage give great relief. These measures can be employed at once in subacute and chronic cases. Diathermy, radiant heat and vigorous massage are the best measures. Massage is often very painful at first, particularly over nodular areas but if persisted in hastens recovery. Injection of a 1 per cent solution of procaine hydrochloride into the several tender areas of the involved structures is a useful procedure. Areas painful to digital pressure are carefully mapped out. A small quantity of procaine is injected into the skin over the site of the lesion, the needle is then inserted until it reaches the tender spot, and 3 cc. of the solution is infiltrated. Following the injection the patient should be encouraged to move and use the part freely. One injection often gives instant and lasting relief. At times it may have to be repeated.

#### PERIARTHRITIS OF THE SHOULDER

Periarthritis of the shoulder, or subdeltoid bursitis, is not restricted to the aged but is commonly seen in men over the age of 50. It is usually associated with injury to the supraspinatus tendon or with calcium deposits in the tendinous cuff that forms the capsule of the shoulder. Bosworth, in a roentgen study of the shoulders of some 6 000 supposedly normal persons, found calcification in these tendons in 27 per cent of all cases. Most of his patients were under the age of 40, but incidence of calcification increased with each decade after 40. Only 3.4 per cent of persons with calcifications had symptoms. Painful shoulders may be without calcifica-

tion Tenosynovitis of the long head of the biceps and its sheath is another cause of painful shoulder

The cause of symptoms is not fully understood The condition is not related to any form of arthritis Trauma seems to be of great importance The original lesion in the supraspinatus tendon may be due to excessive strain, subsequently, sudden wrenching of the shoulder may cause the chalky deposits to initiate an acute irritative reaction

The shoulder syndrome may appear suddenly with intense pain and immobilization of the joint As the acute symptoms subside, the shoulder remains painful, some motion returns but abduction and external rotation are sharply limited Pressure on the tendon of the long head of the biceps may elicit pain There is tenderness on pressure over the shoulder, particularly over its upper and outer aspect, and the patient cannot lie comfortably Sleep may be interfered with for weeks Atrophy of the muscles around the shoulder and trophic disturbances of the hand commonly follow Roentgen examination often reveals calcified deposits in the region of the supraspinatus tendon These subacute symptoms may develop insidiously without a previous acute stage

There is a curious and frequent association of this shoulder affection and angina pectoris With rare exceptions the left shoulder is

ginal pain,

ation Com

immediately following a coronary thrombosis<sup>2</sup>

*Treatment*—In the acute stage, complete rest and immobilization with wet dressings are the best treatment If there is no perceptible improvement within a few days, aspiration

<sup>2</sup> Boas E P and Levy H "Extracardiac Determinants of Site and Radiation of Pain in Angina Pectoris with Special Reference to Shoulder Pain" *Am Heart J*, 14:540, November, 1937

of the bursa and injection with 5 to 10 cc of procaine solution or irrigation of the bursa and washing out of calcified deposits may give quick relief. Roentgen therapy may bring about rapid subsidence of symptoms.<sup>3</sup> A single treatment with a dose of 300 r is often sufficient, but treatments may have to be repeated. Commonly there is an aggravation of symptoms during the first day, with rapid relief from pain and recovery of mobility of the shoulder within 48 hours. Roentgen treatment is less useful in chronic cases, for it benefits only one third of them. Diathermy helps at times, but often gives little relief, and it may intensify the symptoms. Massage should also be employed. In older patients the symptoms may persist for months and cause great suffering. Often codeine must be given to permit sleep. In time there is complete recovery. Frequently the calcified deposits eventually become absorbed.

### NIGHT CRAMPS

Sudden, severe, painful cramps in the muscles of the calf are common in elderly persons. They usually come at night, awaken the patient from sleep, last from a few to 15 minutes and commonly recur during the night. The spastic, contracted muscle is tender and tenderness persists even after the cramp has passed. The patient finds relief by getting up, by massaging the affected part and by applying heat. Their cause is unknown but they occur chiefly in persons who have arthritic changes in the hips or knee joints. They are unrelated to circulatory disturbances in the legs. The blood calcium and phosphorus levels are normal and the administration of calcium does not appear to be useful. Three

<sup>3</sup> Brewer, A. A., and Zink, O. C. "Radiation Therapy of Acute Subdeltoid Bursitis." *J. A. M. A.* 122:800 July 17, 1943.

to 5 gr of quinine taken on retiring by blocking the myo neural junction successfully prevents these cramps. Night cramps may disappear spontaneously for weeks or months but usually recur.

### DUPUYTREN'S CONTRACTURE

Contracture of the palmar fascia occurs not infrequently with advancing age. Its cause is unknown. Certain cases seem to bear a relationship to coronary artery disease with angina pectoris particularly when there is associated peri arthritis of the shoulder. Apparently neurogenic trophic disturbances may be concerned in its genesis. As a rule the condition is bilateral. There is no pain. The slow progressive contracture of the palmar fascia draws the fingers particularly the fourth and fifth into a permanent position of flexion. Treatment is ineffectual, and attempts at surgical correction usually fail.

### OSTEOPOROSIS

With advancing years the bones undergo involutional changes. It is unknown to what extent these are phenomena of normal senescence or to what degree they result from dietary faults or other factors. The average American diet is grossly insufficient in calcium and it is quite probable that this calcium lack accentuated by the passage of years leads to the demineralization of bones that is so characteristic of aged persons. In addition there may be poor absorption of calcium resulting from derangements of digestion of the aged. The end result is osteoporosis with atrophy of the bony cortex, a looser internal architecture and an increasing coarseness of the mesh of the bony trabeculae. Added to this or independently there is osteomalacia, a loss of calcium with softening of the bones. There may be irregular new bone production but the new bone is free from calcium. Roentgen



FIG 34—Senile osteoporosis with compression fracture of the fourth lumbar vertebra



examination reveals the altered structure of the bone as well as its decalcification

As a result of these alterations of bony structure and composition fractures occur more readily most commonly in the spine They are encountered beginning with the seventh decade Low back pain in an elderly person commonly is due to a compression fracture of one or more vertebrae often brought on by minimal trauma In acute cases there may be severe pain and immobility of the back and the development of kyphosis with shortening in stature Any form of low back pain in the aged calls for roentgen examination of the spine which will often reveal a compression fracture Such fractures may be accidental findings in the course of a roentgen examination for another purpose In the lumbar region the intervertebral disks may herniate into the bodies of the softened vertebrae A compression fracture of an osteoporotic spine should be distinguished from one due to metastatic neoplasm A possible primary neoplasm should be searched for The finding of a normal sedimentation rate of the red blood cells and normal values for serum phosphatase helps to exclude the presence of neoplasm

Treatment involves support of the spine with a suitable brace that does not immobilize the patient a diet rich in calcium and supplementary administration of calcium and vitamin D Milk is the best source of calcium and 1 qt should be taken daily In addition calcium should be given in the form of 4 Gm of calcium gluconate or 3 Gm of calcium lactate three times a day Vitamin D is best given in the form of cod liver oil

Postmenopausal osteoporosis is a condition occurring almost exclusively in women who have had a natural or artificial menopause, and is characterized by deficient calcium

deposits in the osteoid tissue.<sup>4</sup> It is a disorder of the bone matrix, not of calcium metabolism. The calcium and phosphorus contents of the blood are normal, but calcium excretion in the urine is increased. The serum phosphatase level is not elevated. There may be kidney stones, due to the increased excretion of calcium.

The spine and pelvis are chiefly involved. The symptomatology is characteristic. A middle aged woman who has passed through the menopause complains of localized pain in the back following a relatively minor jolt or trauma. Roentgen examination discloses a fractured or collapsed vertebra and rarefaction of the bodies of the vertebrae and the pelvic bones.

These patients respond well to treatment with estrogenic hormone. Although there is no primary disturbance in calcium metabolism, it is well to give plenty of calcium in the daily diet—a quart of milk or its equivalent—and vitamin D.

### PAGET'S DISEASE

Paget's disease is a rare condition occurring with equal frequency in men and women past 50. It is characterized by enlargement and thickening of the bones. There are fibrosis of the marrow, irregular bone absorption and much endosteal and periosteal new bone formation, which undergoes irregular calcification. Most of the bones of the body may be involved or the process may be restricted to a few bones, especially the sacrum, pelvis and spine. The serum calcium and phosphorus levels are normal but the serum alkaline phosphatase is much increased (normal is 4–12 King Armstrong units), more than in hyperparathyroidism of similar

<sup>4</sup>Albright F, Smith P H and Richardson A M. Postmenopausal Osteoporosis. J A M A 116:2465 May 31 1941.



FIG 30 - Paget's disease of the skull



FIG 36 Paget's disease of the legs

extent The disease is not caused by altered function of the parathyroid glands

The patient first complains of pain and weakness of the legs The tibiae become thickened, but because of their weakened structure they undergo bowing, with the convexity forward, from the weight of the body Subsequently the femora become involved and thickened and bent with an outward convexity Marked kyphosis of the spine develops, the chest becomes rigid, and breathing becomes largely abdominal The patient grows shorter and his head grows larger Deafness may result from involvement of the petrous portion of the temporal bone, impairment of vision from narrowing of the optic foramina These changes develop very slowly and may remain localized to just a few bones Spontaneous fractures are common, rarely, osteogenic sarcoma develops The disease does not shorten life nor cause death Generalized arteriosclerosis is present in all cases, and recurrent bronchitis is very common, owing to the immobility of the chest

The roentgen picture is quite characteristic The skull is enlarged and greatly thickened and has a moth eaten appearance The bones have a thickened cortex, are poor in calcium, the *normal architecture is largely lost, and they present a spongelike appearance which is due to an irregularity in the distribution of the calcification*

In half the cases, Paget's disease gives rise to no symptoms and is an incidental finding, discovered in the course of roentgen studies for other complaints

There is no effective treatment of Paget's disease Injections of parathormone or of dihydrotachysterol may relieve the bone pains that are often distressing

## METASTATIC NEOPLASM OF THE BONES

Bony metastasis is common to many different neoplasms. It occurs with particular frequency in cancers of the breast, prostate, thyroid and lungs. At times the metastatic lesion gives rise to the very first symptoms of disease and may lead to a pathological fracture or to the erroneous diagnosis of arthritis. Thus a man aged 52 complained of severe pain and limitation of motion of the left hip and of a mild cough. There were clinical signs of an acute lesion of the hip joint, some abnormal physical signs in one lung and a roentgenogram disclosed a neoplasm of the lung. Bronchoscopy verified the diagnosis and roentgen examination of the hip showed metastatic deposits around the hip joint. Another man complained for months of severe pain in the lower thoracic spine. The lower spine was tender and rigid. There were no other physical signs of disease and the roentgen picture of the spine was normal. The pain continued and gradually secondary anemia developed. The sedimentation rate of the red blood cells was persistently rapid. Months after the diagnosis of carcinoma of the pancreas with metastasis to the spine was finally made.

When an older person develops severe bone or joint pain and no other cause is evident, metastatic carcinoma should always be thought of. Often the roentgen examination discloses the nature of the lesion, but at times the roentgen changes may be greatly delayed. A persistently rapid sedimentation rate of the red blood cells, elevation of the serum alkaline phosphatase, low fever, the development of anemia, at times the finding of abnormal white cells in the stained blood smear should suggest the diagnosis. Then a thorough search should be made for the primary lesion. Marked increase in serum acid phosphatase is corroborating evidence.

Treatment can never be curative, but great symptomatic relief often follows high voltage roentgen radiation of the bones involved by the neoplastic process

### MULTIPLE MYELOMA

Multiple myeloma occurs on the average at age 55 and is three times as common in men as in women.<sup>5</sup> It is a neoplasm arising in the bone marrow and invading the bony cortex made up of typical myeloma or plasma cells. Pathologic fractures often occur and may give rise to the first symptoms of disease. Compression fractures of the spine are particularly frequent, and radicular pains are common. Many bones are involved, especially the ribs, spine and skull.

The presenting symptom usually is severe bone pain, most often in the thorax or the lumbar region of the spine. There is tenderness over the affected areas of bone. Secondary anemia becomes marked, with great weakness and progressive loss in weight. Eventually cachexia develops, and uremia is a common terminal event. Bence-Jones protein is found in the urine in about half the cases. There is an increase in both total serum protein and serum globulin contents, an association found in few other conditions. This is probably responsible for the greatly accelerated sedimentation rate of the erythrocytes and of the tendency of the red blood cells to form rouleaux. Abnormalities of the white blood cells are less common, but immature cells of the myeloid series and plasma cells may be found in the blood smear. The blood calcium content is usually normal but may be elevated, the alkaline phosphatase content is normal.

Diagnosis rests on discovery of Bence-Jones protein in the

<sup>5</sup> Bayrd, E. D. and Heck, F. J. 'Multiple Myeloma' *J. A. M. A.* 133: 147, 1947.



37 - Diffuse bone metastasis secondary to carcinoma of the breast





urine and of myeloma cells in aspirated sternal marrow. Roentgen examination reveals typical punched out areas in the bones, especially the skull, ribs and spine. There is no curative treatment. Roentgen therapy may relieve bone pains. Recent reports indicate that a series of 15 intravenous injections of 150 mg of stilbamidine, combined with a diet low in animal proteins, may relieve pain and lead to considerable temporary improvement.<sup>6</sup>

### GOUT

Gout is a metabolic disturbance the true nature of which is not yet understood. For the present we have grouped it with disorders of the joints because its chief manifestations are arthritic and these are often confused with other diseases of the joints. Gout constitutes 5 per cent of all cases of arthritis. It is characterized by an increase of the uric acid in the blood serum, by its familial incidence, by arthritis and by deposition of urate tophi in the joints, bursae, tendons and subcutaneous tissues. In normal persons the level of uric acid in the serum never rises above 5 mg per cent. The gouty person has serum uric acid figures between 6 and 14 mg per cent. High serum uric acid values are found in relatives of persons with clinical gout, who may have no other manifestations of the disease. Similarly high uric acid levels may be encountered in the gouty person long before he has clinical evidences of gout. In this sense we may speak of a gouty diathesis—a familial disorder of uric acid metabolism that underlies and precedes the clinical manifestations of gout. Such a gouty diathesis can be discovered only by chemical examination of the blood and recognition that the

<sup>6</sup>Snapper, I. "Stilbamidine and Pentamidine in Multiple Myeloma." J. A. M. A., 133: 157, 1947.

individual in question belongs to a gouty family. The loose use of the term gouty diathesis and the habit of stigmatizing as gouty, patients with arteriosclerosis, contracted kidneys, psoriasis, eczema or migraine have no basis in fact and lead only to fuzzy thinking and incorrect diagnoses.

It seems probable that a person without the inborn disturbance of uric acid metabolism will not develop gout, but the nature of the dyscrasia is still unsolved. The ancient idea that gout results from overeating and abuse of alcohol must be discarded. Talbott<sup>7</sup> has shown that gouty patients when on a purine free, low protein diet may have acute attacks as frequently as when their food intake is unrestricted. But there are factors that precipitate acute attacks of gouty arthritis, and among these are excessive banqueting and drinking. Some patients discover that the ingestion of some particular article of food will lead to an attack. Injury to a joint or even a mild strain, such as that provoked by a long hike or participation in active sport, may act as the trigger mechanism. An intercurrent infection or a surgical operation commonly brings on acute gout. At times attacks are precipitated by injections of liver extract or of a mercurial diuretic.

Gout is encountered in two forms—acute gouty arthritis, an acute inflammation of one or more joints with constitutional manifestations, and chronic gouty arthritis, chronic joint changes resulting from repeated attacks of acute arthritis with deposition of tophi and permanent joint deformities. Acute gouty arthritis first makes its appearance in the fourth and fifth decades, but many cases are encountered in older persons. Chronic joint changes develop about 10 to 15 years after the onset of gout, so that chronic gouty arthritis

<sup>7</sup>Talbott J. H. *Gout* (New York City: Oxford University Press, 1913)

is seen chiefly after the age of 50 Fully 95 per cent of cases occur in males

In the early years of the disease the pathological changes are restricted to microscopic deposits of urate crystals in the articular cartilages of the affected joints It is this precipitation of urates that is supposed to initiate the acute inflammatory attack of arthritis Gradually through the course of years and following many acute attacks the urate deposits increase and may completely cover the articular cartilage In time the cartilage is destroyed and replaced by urate deposits which may collect in masses forming tophi These changes of the articular surfaces together with the irritative effect of the urate deposits cause a proliferative reaction of the synovial membrane and joint capsule as well as joint and bone changes similar to those seen in osteo arthritis Tophi become deposited in the epiphyseal bone giving rise to the punched out are as visible on the roentgen film In the course of time tophi form in bursae tendons the cartilages of the ears and subcutaneous tissues They may ulcerate when they become very large

The kidneys almost always become affected The earliest findings are urate deposits in the kidney parenchyma In the course of time there is progressive destruction of glomeruli and tubules usually ending with the final picture of primary arteriolar contracted kidney To what extent the pathogenesis of this lesion is related to the urate deposits and to what extent to the hypertension and arteriosclerosis that so commonly accompany gout is unknown Uric acid stones are frequently formed and there may be pyelonephritis

Generalized arteriosclerosis and in particular coronary artery sclerosis are quite constant in advanced cases

Acute gouty arthritis appears suddenly usually between the ages of 35 and 40 More often than not the patient is

awakened from sleep by intense pain in one joint, most frequently the metatarsophalangeal joint of the great toe. By morning the joint is swollen, tense, hot, red and exquisitely tender. There are fever and leukocytosis. The great toe is involved in 50 per cent of cases, but the process may be localized in the ankle, knee or wrist. The shoulders, hips and spine are rarely affected. If untreated, the acute inflammation lasts from three to ten days, usually with nightly exacerbations of pain. In the early stages of the disease tophi are rarely found. With subsidence of the acute inflammatory reaction, the joint returns to normal and the patient may feel better than ever. Within a year or so there is a second attack, and following this the seizures come with gradually increasing frequency.

Chronic gouty arthritis becomes manifest after many years of recurrent attacks of acute gout. In time the inflammation of the joint does not completely subside; some stiffness, pain and deformity remain, and there are acute exacerbations at varying intervals. It is at this stage that tophi appear in the helix and antihelix of the ear, in the tendons or in the subcutaneous tissue around the joints. The olecranon or prepatellar bursae become thickened and filled with urates. Albumin and casts are found in the urine and very gradually evidences of impairment of kidney function appear, marked at first by lowered specific gravity of the urine and lack of concentrating power of the kidneys. Finally there may be kidney insufficiency and eventual death from azotemia. Uric acid calculi may form, and there may be attacks of renal colic. Arteriosclerosis and hypertension with any of their secondary manifestations are common.

*Diagnosis*—Acute monarticular arthritis, developing suddenly, particularly at night in a man aged 40 or over should always arouse the suspicion of gout. If the great toe is in-

involved the diagnosis is practically certain. Characteristic, too, is the fact that the attack subsides in about a week, with complete restoration of the joint to normal. If the patient is seen after he has had a number of attacks, the history is of the utmost importance. The story of repeated attacks of intense monarticular arthritis with a complete remission between attacks is encountered in no other condition. The development of arthritis after one of the known precipitating causes of gout is suggestive. Acute arthritis following operation is almost invariably gouty. The diagnosis of chronic gout is often revealed by the discovery of tophi in the cartilages of the ears or elsewhere or thickened olecranon or prepatellar bursae. A history of former attacks of acute arthritis with complete remissions is very suggestive. Elevation of uric acid in the blood serum to 6 mg per cent or more confirms the diagnosis. The therapeutic test with colchicine is of great value for gouty arthritis alone promptly responds to treatment with this drug. Roentgen studies of the joints may be of assistance. The characteristic finding in chronic gout is sharply demarcated punched out areas in the bone around the joints. Material scraped from a tophus and studied under the microscope will show urate crystals. Such a finding is diagnostic.

The older the patient at the onset of symptoms of gout, the better the prognosis. A patient who has his first attack of arthritis after age 50 does not develop serious pathological lesions. Although the underlying gouty disorder of metabolism cannot be cured, the condition can in large measure be held in check by appropriate treatment.

*Treatment*—If the patient is seen in the acute attack, he should be put to bed and the affected joint protected by a cradle. Hot moist compresses are often soothing. Codeine or morphine may have to be given for the pain. A saline cathartic, such as magnesium sulfate 1 oz., should be given at

once Colchicine is a specific for acute gouty arthritis, although its mode of action is unknown. The active principle is better than the tincture or the wine of colchicum. Colchicine is administered by mouth in tablets of 1/100 gr. Two tablets should be given at once, and following this 1 tablet should be given every two hours until either nausea or diarrhea becomes manifest. With the onset of such symptoms, the drug should be stopped. Relief from pain and diminution of swelling of the joint occur within 24 hours, as a rule. During the acute attack large amounts of fluids should be taken. The diet should be free from purines and low in fats and should consist of milk, eggs, hominy or farina, potatoes, fruits and vegetables such as tomatoes, string beans, cabbage, carrots and beets.\*

When the attack is over, treatment should be continued to prevent recurrent arthritis and the development of chronic gouty arthritis and cardiovascular and renal complications. In spite of the fact that some authors believe that dietary control has no influence on the course of gout, it seems wiser, until we know more about the nature of the disorder, to keep the diet free from purines two days a week and low in purines on the other days. The fat intake should be low, the carbohydrate allowance liberal. Protein should be derived largely from milk, cheese and eggs. Iron and vitamin B complex should be added to the diet. Coffee and tea may be taken, but alcohol should be avoided.

Some patients learn to recognize symptoms of an impending attack, such as twinges of pain in a joint, polyuria, suppression of sweating or gastro intestinal disturbances. In such cases, a saline cathartic and several doses of colchicine 1/100 gr. every few hours may abort the attack. If a gouty

\*Hench P. S. "Diagnosis and Treatment of Gout and Gouty Arthritis" J. A. M. A., 116:453 Feb. 8, 1941

patient contracts an infection or is compelled to undergo operation, the taking of colchicine  $\frac{1}{100}$  gr three times a day may prevent an attack of gout. Some authors advise long-continued intermittent administration of colchicine to patients with severe progressive gout. In chronic gout and in cases with persistent elevation of the blood uric acid level, certain drugs that augment uric acid excretion by the kidneys

TABLE 6 -PURINE FREE DIET

*Foods allowed*

Fruits of all kinds in liberal amounts  
 Vegetables except asparagus, beans kale mushrooms peas spinach  
 Bread and cereals fine cereals like farina, and white bread in limited amounts Avoid whole wheat bread

All meats fish, poultry, game

## SAMPLE MENU

## BREAKFAST

## LUNCHEON

Fruit or fruit juice  
 Farina or rice with cream and sugar  
 Eggs  
 White bread and butter  
 Milk

Cheese eggs or cereal  
 Vegetable salad  
 White bread and butter  
 Fruit  
 Milk

## DINNER

Cream vegetable soup  
 Cheese or egg dish  
 Vegetable potato

Fruit or rice pudding  
 Crackers and butter  
 Milk

should be given. Cinchophen is the most effective of these, but in view of the fact that it may cause acute hepatitis and jaundice it should not be used unless all other measures fail. The salicylates, too, increase the elimination of uric acid. Sixty grains of aspirin should be given daily for four consecutive days of every week. With this, enough alkali should be given to keep the urine alkaline and so prevent the pre-



cipitation of urates in the urine and formation of uric acid gravel or stones. Fifteen to 30 gr of potassium citrate or 30 to 60 gr of sodium bicarbonate should be given three times a day. The patient should be taught to test his urine with litmus paper and to take enough alkali to keep the urine alkaline. Two to 3 qt of fluids should be taken daily.

If cinchophen must be used, it should be given in a dosage of 7½ gr three times a day for three consecutive days every week. The patient should be instructed to report promptly the development of itching of the skin and nausea or other gastro intestinal symptoms. If any such symptoms arise, the drug must be stopped immediately. Fatal liver degeneration may result in persons who are sensitive to cinchophen.

Tophi that become very large or that ulcerate should be excised.<sup>9</sup>

With consistent careful treatment, acute attacks of gout can be avoided, and the development of chronic gouty arthritis and of the chronic visceral manifestations of gout can be prevented.

### FRACTURES OF THE NECK OF THE FEMUR

Fracture through the neck of the femur is a common accident in older persons, one that is attended with grave hazards. In former years, the ensuing prolonged immobility in bed brought with it a mortality of 75 per cent. Laplace<sup>10</sup> reported on 500 fractures of all sorts, excepting skull fractures. Most of them were hip fractures. Of the patients over 80, 52 per cent died, of those between 70 and 79, 35 per cent, of those between 60 and 69, 23 per cent died, and of those

<sup>9</sup>Linton R. R., and Talbott J. H. "Surgical Treatment of Tophaceous Gout," *Ann Surg* 117 161, February, 1943.

<sup>10</sup>Laplace, L. B. "Cardiovascular Complications of Fracture," *Am J Surg*, 44 161, 1939.

under 60, 2 per cent died. An associated cardiovascular condition added greatly to the gravity of the prognosis. Death usually occurred after the first or second week of immobilization and was caused by peripheral circulatory failure (see p 66). The dangers of prolonged bed rest for patients with fractures of the neck of the femur are becoming well recognized. Tinker<sup>11</sup> found that with conservative treatment only 51 per cent of these patients achieved bony union and 25 per cent died. With operative fixation of the fragments, bony union occurred in 78 per cent and the mortality was only 9 per cent. A study of the results of operative treatment by members of the American Academy of Orthopedic Surgeons showed bony union in 88 per cent and a mortality of 8.5 per cent.<sup>12</sup> These figures give telling evidence of the superiority of operative treatment. Operative fixation of the fragments by a nail or similar device and application of an appropriate ambulatory splint within a few days enables the patient to be up and about within a short time and eliminates the hazards of prolonged bed rest.

Many patients can be operated on shortly after the accident. If there is shock, it may be necessary to wait a day or two until this has passed. General anesthesia with cyclopropane or ether is usually well borne. Spinal anesthesia may be used in appropriate cases. The common postoperative complications, in the order of frequency, are infection, pulmonary involvement—pneumonia or infarction, decubitus ulcers, heart failure and coronary thrombosis.

<sup>11</sup>Tinker M. B. and Tinker M. B., Jr. "Fracture of the Neck of the Femur." *J. A. M. A.*, 112:791, 1939.

<sup>12</sup>*Am. Acad. Orthopedic Surg.* "Treatment of Fractures of the Neck of the Femur by Internal Fixation," *J. Bone & Joint Surg.*, 21:485, 1939.

## INFECTIOUS DISEASES

### INFECTIONS OF THE RESPIRATORY TRACT AND EARS

ACUTE tonsillitis is uncommon in the aged, and chronic infections of the tonsil are infrequent as shown by the rarity with which enlarged tonsillar lymph nodes at the angle of the mandible are encountered. Tonsillar tissue diminishes steadily after the sixth decade but rarely disappears completely.<sup>1</sup> In 5 per cent of persons over age 60 the tonsils are large. In the old, as in the young, the tonsils are rarely foci of infection that give rise to constitutional disorders. The arthritides, arthralgias and myalgias are not relieved by tonsillectomy, indeed, this operation is seldom indicated in the aged.

Elderly persons are by no means immune to colds and mild upper respiratory infections, but sinusitis is less common and as a rule milder than in the young. Coryza frequently leads to bronchitis, and bronchitis to bronchopneumonia. Thus every cold should be taken seriously. Bed rest and the maintenance of an equable constant temperature in the room are the most important therapeutic measures. If the nasal discharge or the bronchial secretions become purulent, or if the bronchitis is more than minimal, it is wise to institute im-

<sup>1</sup> Kelemen, G. "Clinical Observations on the Palatine Tonsil in the Aged." *Ann. Otol. Rhin. & Laryng.*, 54:421, June 1945.

mediate treatment with sulfonamides or penicillin to prevent more serious complications such as pneumonia.

Suppurative otitis media is uncommon in the later decades of life. Of 1,514 cases of suppurative otitis media, only 2 per cent occurred in persons over the age of 60.<sup>2</sup> But in these older patients the course of the disease was much more severe. Sixty-eight per cent of those between 60 and 69 and 86 per cent of those between 70 and 79 developed mastoiditis requiring surgical intervention. The evolution of such mastoiditis may be very insidious in older persons, particularly when the infecting organism is pneumococcus type III. It is wise to obtain early cultures of the pus from infected ears to treat such patients vigorously with sulfonamides or penicillin and to keep them under close observation.

### PNEUMONIA

From 6 to 7 per cent of all deaths between the ages of 50 and 80 are caused by influenza and the pneumonias. The death rates remain fairly constant for each succeeding decade; old age does not bring with it a greater chance of dying from pneumonia. But an older person who contracts pneumonia incurs a much greater hazard than a young person, for mortality rapidly mounts with increasing age.

Lobar pneumonia is relatively infrequent among older persons. Of 1,867 cases of pneumococcus pneumonia studied by Cecil Baldwin and Larsen, only 8.7 per cent occurred in persons aged 60 and over. As in the younger age groups, pneumococcus types I, II and III are responsible for fully two thirds of the cases. With advancing years there is a lessening in the occurrence of types I and II and an in-

<sup>2</sup> Rosenwasser, H. Infections in the Nasal Accessory Sinuses and the Ears in Old Age, Arch Otolaryngol 41:182 March 1945.

increasing incidence of type III. The Friedländer bacillus, which is responsible for about 4 per cent of all pneumonias, invades almost exclusively persons past middle age, and attacks males seven times as often as females.

Lobar pneumonia may have the same clinical course in elderly persons as in the young. In the aged and in those greatly debilitated by some chronic disease, it may exhibit such atypical symptomatology that it may not be recognized. The patient may just become confused or stuporous and may develop weakness or paralysis of one side of the body. In such cases the sclerotic cerebral arteries do not supply enough blood to the brain, when toxemia impairs the efficiency of the circulation. Signs and symptoms may be latent, the patient may be up and about and die suddenly, and the pneumonia may be first discovered at autopsy.

Usually there is no initial chill, no chest pain and little febrile reaction. The patient may have had a slight cold and becomes greatly prostrated for no apparent reason. He may complain of headache or may become delirious. In other cases the onset is marked by vomiting or diarrhea and progressive weakness. There is little cough and no expectoration. The temperature may barely reach 100 F. and leukocytosis may be slight. The distinctive feature that should always arouse suspicion of pneumonia is an increase in the respiratory rate. Examination may reveal widespread consolidation of the lungs, out of proportion to the constitutional reaction. When there is much pulmonary emphysema, the physical signs of consolidation may be few and appear late. There may be no dullness, and the development of bronchial breathing is delayed. Defervescence is often by lysis. The mortality of lobar pneumonia is high among older patients and increases with each decade. Pneumococcal endocarditis and meningitis are not uncommon complications.

Bronchopneumonia is a disease of infancy and old age. The majority of pneumonias in older persons are bronchopneumonias. In over 85 per cent of pneumococcus bronchopneumonias, types IV to XXII are the causative organisms. Types I, II and III are rarely encountered. Many other bacteria may give rise to bronchopneumonia. It is a common terminal infection in cachectic patients and in those debilitated by disease. It occurs frequently in patients with pulmonary emphysema and as a sequel to an attack of bronchitis. Aspiration pneumonia develops when, because of impairment of the glottal reflex, mucus and bacteria from the nose and throat are drawn into the lungs. This occurs during anesthesia, coma or with advanced debility. If septic organisms from the teeth or from ulcerating carcinomas of the mouth or upper air passages are aspirated, a necrotizing pneumonia or lung abscess results. Atelectatic pneumonia, a term to be preferred to hypostatic pneumonia, occurs when bronchi become plugged with mucus. Atelectatic areas are produced and these readily become infected. Prolonged bed rest, particularly when the patient is too weak to change position, commonly induces atelectatic pneumonia. Plaster casts that inhibit the respiratory excursions of the thorax act in a similar manner.

Bronchopneumonia usually develops insidiously. It is marked by an increased respiratory rate, variable cough, some febrile reaction and toxemia. Toxemia is manifested by weakness, mental confusion and delirium. The physical findings may be scanty. Usually there is some dullness at one or both

usually absent. In patients with advanced heart disease it is difficult to distinguish between pulmonary infarcts and bronchopneumonia. Both conditions come on insidiously and give

rise to similar symptoms and physical signs. Autopsy studies often indicate that what was interpreted as bronchopneumonia in a patient with advanced chronic heart failure actually was a pulmonary infarct.

Pneumonia in the elderly patient commonly follows a head cold or other upper respiratory infection. The aged should take special measures to avoid such infections and in particular should isolate themselves from friends and relatives who are suffering from colds and coughs. They should avoid going out in inclement weather and exposure to drafts. Persons who are particularly susceptible to winter colds do well in a southern climate. They should avoid large populous centers like Miami, where fresh infections are brought in daily by the thousands of visitors from the North, and settle in a small, quiet community. To avoid pneumonia, every effort should be made to prevent old people from becoming bedridden. When they are confined to bed, their position should be constantly changed and they should be propped up as much as possible. Plaster casts that immobilize the chest should be avoided. Codeine and morphine should be used sparingly for they suppress the cough reflex and favor the development of atelectatic pneumonia. Recent studies hold out some promise of successful prophylactic vaccination against pneumonia. Kaufman<sup>3</sup> has reported vaccination of several thousand inmates of the New York City Home with a capsular antigenic polysaccharide. Among the vaccinated group in two successive winters, there were only one-quarter as many cases of pneumonia as in the nonvaccinated controls. If these observations are confirmed, vaccination may prove a valuable measure against upper respiratory infection in susceptible persons, and a prophylactic against postoperative pneumonia.

<sup>3</sup>Kaufman P. "Studies on Old Age Pneumonia," *Arch. Int. Med.* 6: 201 February, 1941.

*Treatment*—If treated early, the pneumonias of older persons respond well to the sulfonamide drugs and to penicillin. Chemotherapy has greatly brightened the outlook in this serious infection of the aged, which formerly brought with it a mortality of over 50 per cent. The first element in successful treatment is early diagnosis. If pneumonia is recognized within 24 hours of its onset, it usually responds promptly to chemotherapeutic treatment.

The general treatment of pneumonia corresponds to that given pneumonia patients irrespective of age. The fluid intake should be liberal and the diet light. Oxygen is administered if there is much cyanosis or labored breathing. These aging patients have not the endurance of younger patients and should be spared every unnecessary strain. Supportive treatment of the heart is often necessary. In the young we no longer give digitalis routinely in case of pneumonia. But in the elderly and aged, who so often have some complicating cardiovascular disease or who have advanced pulmonary emphysema which increases the work of the right heart, digitalis is often helpful and at times life saving. In the presence of hypertension and advanced coronary sclerosis or of pulmonary emphysema, digitalis should be given. If the patient has not been taking digitalis,  $1\frac{1}{2}$  gr. should be given three times a day for the first three days, and after that twice a day for another week.

Enough codeine, morphine or other sedatives may be used to ease the cough and allay restlessness, but, as mentioned previously, extreme caution is necessary in using these drugs.

### ERYSIPELAS

Erysipelas is more common in persons of advanced years than in persons in the younger age groups. Old people are



apt to become careless about the cleanliness of their bodies their skin is dry and atrophic and minor abrasions and cracks of the skin occur and are easily overlooked, with resultant infection. Debilitating chronic diseases diminish resistance to infection. Two thirds of the cases start on the face, the rest mostly on the legs. Often the clinical course resembles that seen in younger patients, but in many instances, just as in the case of the pneumonias the disease comes on insidiously without great febrile reaction. The mortality in the past has been high over 40 per cent. Since the introduction of the sulfonamides the outlook has been greatly improved. As soon as the diagnosis has been made, 2 to 4 Gm of the drug should be given immediately. Then 1 Gm should be given every three hours day and night. Usually the temperature drops and the progress of the lesion is arrested within 24 hours. The dosage should be continued at 1 Gm every four hours for another day or two until it is certain that the infection is clearing up. It may be well if there is appreciable residual infection, to continue with 1 Gm two or three times daily for a few days longer. Penicillin is equally effective. Exposure of the affected area to ultraviolet radiation usually arrests the progress of the lesion within 24 to 48 hours.

### SYPHILIS

This is no place for a detailed discussion of syphilis and its complex symptomatology. Syphilis is a disease of youth and even its tertiary and neurological manifestations develop for the most part, before the age of 50. If patients with syphilis survive beyond the sixth decade, their symptoms and the indications for their treatment do not differ from those of the younger age groups. Cardiovascular syphilis is discussed in Chapter 7. Primary or secondary syphilis oc-

curing in older persons runs the same course as in the young and calls for the same treatment

*General paresis*—About 10 per cent of paretics admitted for the first time to state hospitals in New York State are over the age of 60. Four times as many males as females are admitted. The incubation period of paresis is the shorter, the older the patient when infected. Therefore, in paretics 60 years of age or older the primary infection has taken place either a long time ago (40 or 50 years) or recently (within five to seven years). The symptoms are those of an organic psychosis with no distinguishing characteristics. There are impaired memory and judgment, senile deterioration with delusional trends. The neurological signs are difficult to distinguish from those of cerebral arteriosclerosis. The Argyll Robertson pupil is by no means constant. The blood Wassermann reaction is negative in about 30 per cent of cases in contradistinction to the invariable positive reaction in the younger age groups. The Wassermann reaction and the colloidal gold curve of the spinal fluid are positive. This indicates the need for examination of the spinal fluid of all older psychotic patients to avoid missing the diagnosis.\*

*Latent syphilis*—The chief problem that arises in older persons is posed by the discovery of a positive Wassermann reaction in an old person. The diagnosis of syphilis in old persons is difficult because the clinical picture is not so typical as in younger persons. The diagnosis should be made with particular attention to the cardiovascular and nervous systems.

In every case a spinal tap should be made and a complete examination of the spinal fluid, including particularly the cell count, the Wassermann reaction and the colloidal gold

\* Arieti S. General Paresis in Senility. Am J Psychiat 101:585 March 1945

test should be carried out. If there is evidence of visceral syphilis or if the spinal fluid shows a positive reaction the case should not be classed as one of latent syphilis, and active treatment should be given. Only if the spinal fluid is normal and there are no physical signs of organic disease due to syphilis should the diagnosis of latent syphilis be made.

Latent syphilis encountered less than four years after the initial infection calls for continuous active treatment as given for early syphilis. The management of latent syphilis discovered more than four years after the initial lesion should be individualized. If the patient is so old that his life expectancy is only 10 to 15 years, the hazards of treatment with arsenicals may be greater than the risk of the development of syphilitic lesions. Moreover, there is such a thing as spontaneous cure of syphilis, and arrest of the disease is not uncommon. As a rule, if latent syphilis is accidentally discovered in a person over the age of 60, it is wisest to withhold treatment unless a full course of penicillin is given, as outlined below. It is particularly dangerous to give a few casual injections of arsenic or bismuth to such patients, for this may disturb the immunity relations between the spirochete and the body and stir up the disease.

If the spinal fluid reaction is negative in a patient with latent syphilis at least four years after the initial infection there is no need to worry about the possible subsequent development of neurosyphilis; it practically never occurs. But if the spinal fluid gives any indication of syphilis even if the patient is asymptomatic, and the blood Wassermann reaction is negative, active treatment must be instituted because in these cases there is grave danger of the future development of neurosyphilis. Such patients should be treated as are those with neurosyphilis at younger ages, and this in

volves the use of tryparsamide and of fever therapy if the simpler methods do not bring results

In a patient over the age of 60 with evidences of visceral or cerebrospinal syphilis enough treatment should be given to bring about symptomatic relief it should not be pushed as vigorously as in a younger person Tabes dorsalis may be arrested and inactive in a patient past 60 In such cases treatment may be unnecessary and inadvisable

The treatment of syphilis and of neurosyphilis is in a state of flux pending evaluation of penicillin therapy Preliminary reports indicate that penicillin will be a most valuable aid in the treatment of latent syphilis and of neurosyphilis Final opinion on the optimal dosage has not crystallized The average course of treatment for neurosyphilis is 80 000 units intramuscularly every three hours day and night for 7½ days or a total of 4 800 000 units<sup>5</sup> Such dosage may be repeated one or two times if there is no appreciable improvement but this is rarely necessary Most of the improvement occurs in the first four months but may continue to become manifest for a year or more A favorable change in the spinal fluid occurs in about 74 per cent and normal spinal fluids result in 36 per cent Clinical improvement is observed in 65 per cent of patients with symptomatic neurosyphilis and in 30 per cent of those with paresis or with tabes dorsalis In asymptomatic neurosyphilis penicillin is more effective than any other measure in reducing the spinal fluid to normal Such treatment would appear to be suitable for latent syphilis as well

The old line therapy for latent syphilis involves three courses of eight intramuscular injections of 3 gr of bismuth in oil alternating with three courses of eight intravenous in

<sup>5</sup>Stokes J H and Steger H P "Penicillin Alone in Neurosyphilis"  
J A M A 131:1 May 4 1948

*THE PATIENT PAST FIFTY*

jections of neoarsphenamine During the following two years the patient should receive each year two courses of 12 injections of bismuth No attempt should be made to push treatment to achieve a reversal of the positive Wassermann reaction This often cannot be accomplished in these late cases and is clinically unimportant

## Chapter Sixteen

### DIABETES

DIABETES has become a problem of middle age and of the later years of life. It is estimated that there are 500,000 diabetics in the United States.<sup>1</sup> Of these, one quarter are under the age of 50, one-quarter are between 50 and 60 and one half are over the age of 60. Yet only one ninth of the population is over 60 years old. The age-specific prevalence rates of diabetes rise steadily with the years; the rate of increase is accelerated after age 50 up to age 75, when the incidence

is more rapidly than the general population. The increasing numbers of old people and of women in the population. The onset of the disease occurs under age 50 in one fourth of the cases, between the ages of 50 and 64 in one-half and over age 64 in one-quarter. The average age at onset is 54 years. As a result of modern methods of treatment, diabetics are spared death from coma and survive to die in their later years from some complication. In older persons diabetes appears in a milder form and many older diabetics have had the disease for years. The average duration of life

<sup>1</sup> Spiegelman, M. and Marks, H. H. "Age and Sex Variations in the Prevalence and Onset of Diabetes Mellitus," *Am J Pub Health* 36:28, January, 1946.

when diabetes has had its onset at age 50 is 14 years, at age 60 it is 10 years, and at age 65 it is 8 years. The average age at death is about 64 years, and most diabetic deaths occur during the seventh decade of life.

The causes of death of diabetics have undergone a radical change during the past 40 years. Today the cardiovascular renal diseases are the leading causes, accounting according to Joslin's figures, for some 60 per cent of deaths. Breaking down this figure further, we find that cardiac disorders account for 30 per cent, cerebral vascular insults for 11 per cent and gangrene of the extremities for 4 per cent of deaths. The average age at death from an arteriosclerotic complication is 68 years. Cancer is responsible for about 8 per cent and tuberculosis for about 4 per cent of deaths.

In the old, as in the young, hereditary influences and obesity favor development of the disease.

In elderly persons the onset of diabetes is usually insidious. Most often it is evidenced by polyuria, loss of weight and pruritus vulvae. Commonly it is discovered by routine urine examination of a patient who comes to the physician with another complaint. Arteriosclerotic heart disease or peripheral vascular disease often brings the unrecognized elderly diabetic to the physician. Hypertension is encountered in fully one half of all older diabetics.

### MANAGEMENT

General treatment of the elderly diabetic corresponds to that of younger diabetics. The urine must be kept sugar free and the weight of the patient held within average normal figures. In recent years there has been an increasing tendency to give more sugar and less fat in the average diet. This seems particularly important in older patients because

of the apparent relationship between fat and cholesterol metabolism and atherosclerosis. The average dietary should contain in the neighborhood of 150 Gm of carbohydrates, 80 Gm of proteins, and 100 Gm of fat. Exercise increases the sugar tolerance of diabetics, and all patients should be encouraged to engage in moderate physical activity up to their capacity. The older the patient, the more slowly should treatment be instituted. It is not wise to make an elderly patient undergo strict starvation at the onset of treatment in an endeavor quickly to free his urine from sugar and reduce the level of sugar in the blood. Such radical treatment favors the development of thromboses in the coronary, cerebral or peripheral arteries. It is much better to proceed slowly and to allow several weeks for establishment of proper dietary adjustment, and for elimination of glycosuria and hyperglycemia.

In many old patients, mild diabetes can be controlled without insulin. If glycosuria does not yield to dietary measures alone and if the patient continues to lose weight insulin should be given. Most cases can be controlled with an appropriate dose of protamine zinc insulin. For details, the reader should consult any of the diabetic handbooks.

Special care should be taken to avoid insulin overdosage and shock because in older persons hypoglycemia often leads to vascular accidents in the domain of the cerebral, coronary or peripheral arteries. Insulin administration should be preceded by careful dietary control over a period of time, so that the approximate insulin needs of the patient can be estimated. The initial dose of insulin should be small and the amount gradually increased. If protamine zinc insulin is

A major part of the treatment of the elderly diabetic con-



cerns itself with the prevention early recognition and therapy of complications

*Arteriosclerosis* develops with much greater frequency and at younger ages in diabetics than in the population at large. Men and women are affected alike so that diabetic women in contrast to women without this disorder have arteriosclerosis of the coronary cerebral and peripheral arteries as commonly as do diabetic men.

Arteriosclerosis of the coronary arteries giving rise to the syndromes of angina pectoris and cardiac infarction is a major problem in elderly diabetics. The treatment of this heart condition in diabetics does not differ from that of patients with normal sugar metabolism. The diabetes should be treated conservatively. Although the urine should be kept free from sugar no rigorous attempts should be made to reduce the blood sugar levels to normal figures. These patients do not stand too rigid dieting and do best on a diet containing about 150 Gm of carbohydrate. Too great restriction of carbohydrate may result in frequent anginal attacks which cease when the diet is again made more liberal. Protamine zinc insulin or regular insulin may be used freely in these patients but sudden large increases in dosage should be avoided and great care should be taken to avoid hypoglycemia and hypoglycemic shock. Such an event may induce cardiac infarction.

Peripheral arteriosclerosis is almost universal in long standing diabetes. Calcification of arteries can be demonstrated in 90 per cent of patients who have had diabetes for 10 years or more. This is true even in the young. Arterial occlusion with resultant gangrene is common and its frequency increases with age. The average age at onset is 64. Gangrene develops predominantly during the winter months. Prevention of gangrene and infection of the lower extremities

involves the same principles that have been outlined in the discussion of peripheral arteriosclerosis (p 136) There must be scrupulous cleanliness of the feet and avoidance of trauma The feet should be kept warm in cold weather The diabetes too, should be well controlled If dry gangrene develops, conservative treatment is indicated These patients have a short life expectancy and amputation involves a serious hazard Often it reduces them to a state of invalidism, and they never learn to walk with an artificial limb Commonly gangrene in the other leg starts soon after amputation of the one first affected Many of these patients may come through with only the loss of all or a part of a toe if they are treated with infinite patience and meticulous care The patient should be kept in bed for activity of the muscles greatly increases their need for blood, and the narrowed arteries cannot admit enough flow to supply the muscles as well as the feet The feet should be kept below heart level so that the force of gravity may aid in the flow of blood to the extremity The foot should be protected by a cradle dressings, as a rule are unnecessary The application of heat is dangerous unless the temperature is rigidly controlled If the temperature is too high, the local metabolism of the tissues is increased, and this calls for more blood than the diseased arteries can supply Pain often results and there may be an extension of tissue destruction If the temperature is too low it may induce vasospasms and further impair the local circulation The temperature of the extremity should be kept constant at 92 F by means of a thermostatically controlled heating device in the cradle The only operative procedure permissible is simple drainage, if necessary, proximal to the inflammatory area of the zone of demarcation One should wait until spontaneous sloughing takes place, and this may take weeks or months

When there is moist gangrene, the situation becomes much more urgent, for then there is active infection of the tissues. Wet dressings should be applied and full doses of sulfadiazine or penicillin given. The diabetes should be carefully watched, for with such infection the diabetes rapidly becomes more severe and acidosis readily occurs. The carbohydrate intake and the amount of insulin should be increased. If the infection does not show prompt signs of recession, early amputation should be undertaken.

Before amputation careful studies of the arterial circulation of the leg should be made. The oscillometer and studies of skin temperatures give the best information. Amputation should be done below or above the knee depending on circumstances. Lower amputations are rarely indicated. Anesthesia is best given by the spinal route. Treatment of the diabetes immediately before and after operation corresponds to that outlined subsequently for all operations or infections.

*Furuncles* and *carbuncles* constitute serious complications. The patient should at once receive full doses of sulfadiazine or penicillin. Radiotherapy should be given to the carbuncle simultaneously. If the carbuncle continues to develop despite treatment and if the diabetes begins to get out of control, operation with excision of the carbuncle may become necessary. Carbuncles are frequently followed by cortical abscesses of the kidneys and by perinephric abscess. This contingency should always be kept in mind.

*Pneumonia* complicating diabetes is treated as it is in other patients.

In the presence of *intercurrent infection*, whether it be carbuncle, pneumonia, an infection of the leg or of the kidney, adjustment of the diet and of insulin must be made. The diet should contain a liberal portion of carbohydrates and should consist of milk, cereal, cream and eggs. The urine should be

examined every few hours. Insulin is given every three or four hours in a dosage adjusted to the amount of sugar in the urine. Regular insulin should be used. A rough and ready guide is the color of the Benedict solution after reduction. If the color is red, give 20 units of insulin, if it is orange, give 15 units, if yellow, 10 units, and if yellowish green, 5 units. When the infection has subsided and the temperature has returned to normal the regular diabetic diet with appropriate insulin dosage can be resumed.

Operation and anesthesia induce a serious disturbance of carbohydrate metabolism, probably by damaging liver function, in particular the storage of glycogen. Before operation, the patient should receive from 150 to 200 Gm of carbohydrate a day for several days and enough insulin to assure its utilization. In case of an emergency operation it is well to give 100 Gm of carbohydrate intravenously before proceeding. Following operation at least 150 Gm of carbohydrate should be given daily, intravenously if necessary. On the morning of operation one half to two thirds of the usual dose of protamine zinc insulin should be given and this may be continued after operation if the intake of carbohydrate is kept adequate. After operation crystalline insulin should be given every four hours in dosage determined by the urinary reaction for sugar, in the same manner as is done in case of infections. Spinal anesthesia and ethylene seem to be the anesthetics of choice for diabetics. General anesthetics often cause serious disturbance of liver function and so interfere with carbohydrate metabolism by preventing storage of glycogen in the liver. One third of all operations on diabetics are amputations. The operative mortality is about 11 per cent. Carbuncles have an operative mortality of 9 per cent. Infections of the urinary tract are common in diabetics. Autopsy studies reveal that about 20 per cent of persons

dying of diabetes have infection of the kidneys. Cortical abscesses of the kidneys have been mentioned. Ascending infections of the urinary tract are of frequent occurrence, particularly when there is some obstruction in the urinary tract. Such infections may be asymptomatic and discoverable only by finding bacteria and pus in the urine. They may suddenly light up and give rise to a severe necrotizing pyelonephritis. An unexplained increase in the severity of the diabetes always calls for investigation of the urinary tract. Patients with prostatic enlargement should be handled with particular care. Catheterization should be avoided unless absolutely necessary and then should be carried out with meticulous asepsis. Just as in nondiabetics, urinary infections are often controlled by the administration of sulfadiazine, 1 Gm. four times a day, or of streptomycin. The diabetes should be treated vigorously, and plenty of fluid should be given. As soon as possible, a complete investigation of the urinary tract should be undertaken.

*Intercapillary glomerulosclerosis* is the name given to a characteristic glomerular lesion found in approximately two thirds of all diabetics at autopsy, especially in those over the age of 40.<sup>2</sup> There is a focal or diffuse fibrosis of the glomeruli with scattered typical spherical hyaline lesions. Similar lesions occur in about 12 per cent of patients with subacute or chronic glomerulonephritis, but otherwise are rarely encountered in nondiabetics. The development of the lesion bears no relationship to the degree or duration of the diabetes nor is it necessarily accompanied by hypertension, albuminuria, renal insufficiency or edema. Kimmelstiel and Wilson,<sup>3</sup>

<sup>2</sup>Laipply, T. C. et al. "Intercapillary Glomerulosclerosis." Arch. Int. Med. 74:354, 1944.

<sup>3</sup>Kimmelstiel, P. and Wilson, C. "Intercapillary Lesions in the Glomeruli of the Kidney," Am. J. Path., 12:83, November, 1936.

in their original description, associated a distinct clinical syndrome with the lesion, namely, diabetes, marked albuminuria extensive edema and frequently hypertension and renal insufficiency. More recent studies show that only 6 per cent of persons in whom intercapillary glomerulosclerosis is found at autopsy show this clinical picture. When it occurs the syndrome runs the course of the nephrotic stage of chronic glomerulonephritis with a poor prognosis.

Gall stones occur with particular frequency in diabetics. Joslin reports that at autopsy gall stones are found in fully 25 per cent of diabetics, as contrasted with at most 10 per cent in nondiabetics. Many of these patients have no symptoms of gallbladder disease. When symptoms do occur they usually antedate the diabetes by about five years. It is a moot question whether chronic gallbladder infection can cause diabetes but it certainly can make it more active. Gall bladder disease in diabetics is dangerous because complications such as cholecystitis and cholangitis are not only serious in themselves but may induce an increase in severity of the diabetes. Operation for gallbladder complications in diabetics carries a mortality of some 10 per cent. It is wise therefore, to remove a gallbladder containing stones from a diabetic as soon as it is recognized, before more serious complications may develop.

When *appendicitis* occurs in a diabetic the onset is apt to be insidious and the symptoms atypical. They may simulate diabetic coma. Appendicitis carries an operative mortality of 10 per cent in diabetics. Lowering of this rate depends on constant alertness and vigilance on the part of physicians.

Cancer occurs not infrequently in diabetics. Curiously enough cancer of the pancreas is especially common comprising fully 10 per cent of all cancers encountered in diabetics. It does not seem to aggravate the severity of the diabetes.

*Tuberculosis* occurs with sufficient frequency in diabetics to make it necessary to look for it in every patient with pulmonary symptoms or unexplained fever. In such cases a roentgen film of the chest and a sputum examination are indispensable. The presence of diabetes accelerates the course of pulmonary tuberculosis unless the diabetes is vigorously treated.

*Cataracts* of the senile type are common in elderly diabetics and may require operation. Initial treatment of diabetes is often associated with a reduction of near vision. The cause of this hyperopia is not fully understood but it seems to depend on changes in refraction of the lens. It disappears when the diabetes is brought fully under control. In the interim the patient may have to wear spectacles with plus 3 lenses. Tobacco amblyopia seems to be more common in diabetics than in persons with normal sugar metabolism. A diabetic who complains of disturbances of vision should give up smoking.

The nature of so called *diabetic retinitis* is not yet clearly understood. Diabetic retinitis is characterized by tiny punctate hemorrhages and deep lying circular punctate exudates with sharp margins usually distributed around the macular region. The retinal veins may be dilated and there may be nodular dilatations and constrictions of the veins which are usually associated with large areas of hemorrhage. There may be hemorrhage into the vitreous and subsequent scar formation and detachment of the retina. The retinal lesions bear no relationship to the severity of the diabetes nor to the degree of care with which it has been treated but they are uncommon in cases of less than three years duration. Diabetic retinitis may develop in the absence of hypertension or vascular disease. It can be distinguished from hypertensive retinitis whose features are flame-like hemorrhages and fuzzy

cotton wool exudates. When the two conditions occur simultaneously it may be difficult to differentiate one from the other. The prognosis of diabetic retinitis is better than that of retinitis associated with hypertension. Vision usually remains good unless the macula is involved. Wagener and his associates<sup>4</sup> encountered diabetic retinitis in 17.7 per cent of 1,052 cases of diabetes. It occurred chiefly in patients over the age of 40 and was often accompanied by hypertension. In 25 per cent it was associated with diabetic neuropathy.

*Diabetic neuropathy* occurs in patients with neglected and poorly treated diabetes. In them it may be precipitated by an intercurrent infection, an operation or a bout of diabetic coma. Neuropathy occurs as often in mild as in severe cases of diabetes. It is caused apparently by a basic metabolic disturbance induced by the diabetes and not by a vitamin B deficiency.<sup>5</sup> The neuropathy may involve not alone the peripheral sensory and motor neurons but elements of the autonomic nervous system concerned with intestinal, bladder and vasomotor functions. Early symptoms are usually manifested in the lower extremities and are characterized by muscular weakness, pains and cramps worse at night and on exposure to cold. The tendon reflexes may be diminished or absent and there may be diminution of cutaneous sensations and of vibratory sense. Muscle tenderness is common. Impairment of peripheral sympathetic nerve supply may give rise to sweating deficiencies, loss of vasomotor control and dependent edema and trophic disorders of the skin. The pupils may be sluggish, small, irregular and unequal. Anorexia, nausea, obstinate constipation or constipation with alternating diarrhea may occur. In

<sup>4</sup>Wagener H. P. et al. "Retinitis in Diabetes." New England J. Med. 111: 1131, 1934.

<sup>5</sup>Rundles R. W. "Diabetic Neuropathy." Medicine 24: 111, May, 1945.



potence and true atonic bladder paralysis are not uncommon. Orthostatic hypotension has been observed. The spinal fluid often shows moderate elevation of total protein, which in severe cases may rise to levels as high as 400 mg per cent.

The diffuse nerve lesions and the abnormality of the spinal fluid indicate that diabetic neuropathy is caused by a systemic disorder, namely, the disturbed metabolism of long-standing unregulated diabetes. This view is confirmed by the fact that the only effective treatment consists of strict regulation of the diabetes and the use of insulin. The administration of vitamins is of no benefit. Early diagnosis is important, for if promptly and adequately treated the neuropathic changes are reversible.

## DISEASES OF THE THYROID GLAND

### HYPERTHYROIDISM

**HYPERTHYROIDISM** in the aged does not manifest the classic symptoms of exophthalmic goiter and consequently is often overlooked. An unexplained loss of weight or a rapid pulse rate in an older person should always arouse the suspicion of hyperthyroidism. Indeed these may be the only symptoms. Usually marked asthenia, sleeplessness and increased nervousness are also present. Commonly the patient does not present the marked motor restlessness that is so characteristic of younger patients. Some are so quiet and depressed that the condition has been designated the apathetic type of hyperthyroidism. Enlargement of the thyroid gland and abnormal eye signs are commonly lacking.

These patients present themselves to the physician in various guises. One group complains of loss of weight and

the patient appears wasted. In some even cachectic and usually the suspicion of carcinoma is aroused. The distinctive feature that should always suggest hyperthyroidism is the rapid pulse rate ranging between 100 and 120. Yet there are some patients whose pulse rates never exceed 80. At times

auricular fibrillation may lead to the correct diagnosis. The blood cholesterol is usually low. Careful inquiry into the symptomatology will usually reveal increased nervousness and insomnia, some shakiness of the hands, a tendency to diarrhea and intolerance of heat.

More rarely abdominal symptoms dominate the clinical picture. There may be persistent nausea, vomiting and abdominal pain. More commonly diarrhea manifests itself. This may be so severe as to suggest the existence of ulcerative colitis. Again the rapid pulse rate and marked loss of weight should direct attention to the thyroid gland. The diagnosis is confirmed if gastro-intestinal studies are negative and a basal metabolic rate determination reveals an elevated metabolism.

Usually a cardiac symptomatology is so much in the foreground that the patient is suspected of having some primary form of organic heart disease. The absence of thyroid enlargement or abnormal eye signs and maybe of tremor makes it easy to overlook a thyroid disorder. The patient complains of dyspnea and palpitation. He has lost weight and may give a history of nervousness and diarrhea. He may be intolerant of hot surroundings. On examination the only significant finding may be a heart rate between 100 and 140 or auricular fibrillation with a rapid ventricular rate. Auricular fibrillation in the absence of demonstrable organic heart disease in older people should always arouse the suspicion of hyperthyroidism. The following case may serve as a typical example.

A man aged 60 for two years had had increasing weakness, dyspnea on exertion and palpitation. For one month the symptoms had been worse. His weight one year before was 193 lb., his present weight 150 lb. He was a serene, well built man. The eyes were not prominent. The thyroid gland was not palpable and no substernal struma was visible on the roentgen film. The lungs were emphysematous, with

some rales at the right base. The heart was not enlarged, the sounds were rather faint and there were no murmurs. The rhythm was absolutely irregular, with a ventricular rate of 150. The electrocardiogram showed auricular fibrillation. The blood pressure was 140/80 mm. The radial and temporal arteries were sclerotic. It was thought that the patient had auricular fibrillation associated with arteriosclerosis. There was no other evident etiological factor. Digitalis was administered. Twenty-one cubic centimeters of the tincture was given in three days, yet the ventricular rate was 120. In the first nine days of treatment 46 cc. of the tincture was administered, there was no nausea or bogemmy and the ventricular rate did not fall below 90. In the subsequent seven days 39 cc. more of tincture of digitalis was given with no further drop in pulse rate. The unexplained resistance to the effects of digitalis led to estimation of the patient's basal metabolic rate. This was plus 54 on one occasion and plus 65 on another.

Cardiac symptoms in hyperthyroidism are conditioned by various physiological disturbances of the circulation which serve to increase the work of the heart to such an extent that cardiac enlargement, and eventually heart failure, may ensue. The factors increasing the work of the heart are the increased metabolism, necessitating an increased minute volume flow of blood, an increased blood velocity and an increased blood volume, and the direct effect of the thyroid secretion on the heart. When there is pre-existing heart disease, cardiac disability develops much more rapidly. Congestive failure occurs chiefly in patients with some form of organic heart disease. The extra load superimposed by the thyroid disturbance overtaxes the heart, whose reserve is already impaired.

Because of the high incidence of cardiac disorders in older people, cardiac complications occur much more frequently in older individuals with hyperthyroidism than in the young. In the earlier stages, before serious damage to the heart has developed, the symptoms are those arising from an overactive

heart and an unstable vasomotor mechanism, registered by a hypersensitive nervous system. The patient complains of palpitation, pulsations in the neck, head and epigastrium and shortness of breath. The pulse rate is rapid and is little slowed by rest or sleep. The heart may or may not be enlarged. Enlargement is more common in older individuals and in those with auricular fibrillation. The first heart sound is sharp and snapping and may simulate the one heard in mitral stenosis. The electrocardiogram is not characteristic. The systolic blood pressure is usually elevated from 10 to 30 mm Hg and is extremely labile. If the diastolic pressure is over 95, there is a concomitant essential hypertension.

Auricular fibrillation occurs during the course of hyperthyroidism in about 20 per cent of all cases. Its incidence is much greater in the older age groups. At first it is paroxysmal in its appearance. The attack may last minutes, hours or days, and between the paroxysms the rhythm of the heart is regular. The patient experiences marked palpitation and some dyspnea during the attack but characteristically is not nearly as disabled as the patient with auricular fibrillation associated with mitral stenosis or hypertension. In spite of a rapid ventricular rate he usually walks about with little cardiac distress and without evidence of congestive heart failure. In the course of time the irregularity comes on more and more frequently and the duration of the attacks is longer, until finally auricular fibrillation becomes permanently established. Even at this time heart failure occurs only in very long-standing cases or when there are complicating cardiac lesions. Auricular fibrillation of hyperthyroidism is marked by rapid ventricular rates. Another characteristic is the resistance of the ventricular rate to slowing by digitalis. Exceptionally large doses must be given to achieve this effect, and such doses are tolerated without the appearance of toxic

symptoms This large tolerance for digitalis may at times have diagnostic value disclosing on further investigation an occult case of hyperthyroidism

When hyperthyroidism is superimposed on valvular or hypertensive heart disease its recognition is still more difficult Tachycardia out of proportion to the cardiac lesion an unexplained auricular fibrillation or loss of weight should lead the physician to check the patient's basal metabolic rate The added strain imposed by the hyperthyroidism may in such cases give rise to serious cardiac symptoms Patients with enlarged hearts may develop heart failure and those with coronary arteriosclerosis may experience frequent anginal attacks

*Treatment*—As in the young successful treatment usually calls for operation Older persons resist the idea of operation and of course they do present a greater operative risk When ever restoration of health by subtotal thyroidectomy seems possible surgical intervention is the treatment of choice If the patient refuses medical treatment is instituted The patient should be put to bed and given a high caloric diet He should be relieved of as many cares and responsibilities as possible Three to five drops of Lugol's solution should be given once a day Sedatives such as phenobarbital should be freely administered If the patient gains weight and if the pulse rate slows and the basal metabolic rate drops the patient may gradually be allowed more liberties However the iodine medication and a modified rest cure should be continued for six to twelve months for the arrest of active hyperthyroidism is a slow process and cure is rare Exacerbations of symptoms may occur at any time even during careful treatment

Thiouracil is useful in the treatment of some older persons for whom operation is not feasible Thiouracil leads

to a fall in oxygen consumption and in basal metabolic rate by inhibiting the production of thyroid hormone. The thyroid gland becomes hyperplastic but contains no colloid. As a result of the administration of thiouracil, the symptoms of thyroid intoxication disappear within a few weeks and remain in abeyance as long as the drug is given. Thiouracil does not cure hyperthyroidism, for when its administration is stopped excess hormone is again produced and the original symptoms recur. However, hyperthyroidism has a cyclic course, and spontaneous remissions occur. It may be possible to give the drug until such a spontaneous remission takes place. It is not without danger, for a few fatalities from agranulocytosis have been reported. The dosage is 0.2 Gm. two or three times a day for four weeks, then 0.2 Gm. daily for six months. White blood cell counts should be made twice a week to detect the earliest evidence of granulopenia. If the white cell count drops appreciably, the drug should be stopped at once. Some patients develop a transient rash or fever. This is not of serious import and does not call for stopping the drug.

Subtotal thyroidectomy is the treatment of choice, because it offers the most favorable prospect for quick and permanent cure. An added indication in patients with adenomatous goiter is the danger, with advancing years, of malignant degeneration of the gland. With meticulous preoperative preparation and skillful operation the risk is small. Preparation for operation consists of bed rest and the administration of a high caloric diet and 10 drops of Lugol's solution three times a day. Sedatives should be given as needed. Preoperative cure may take one week or many weeks. Most patients can be brought to an optimum condition within two weeks. But operation should never be hurried. A favorable response is evident in a gain in weight and a reduction of pulse rate.

and basal metabolic rate. A drop of metabolic rate to plus 30 or below indicates that it is safe to operate. If the patient does not respond to preoperative treatment, operation should be postponed. In borderline cases the two stage operation should be performed in order to maintain complete control.

The presence of a cardiac complication, whether it be simple enlargement, auricular fibrillation or congestive heart failure, is an indication for early operation. Only by such a radical measure can one hope to avert further cardiac damage and to restore normal cardiac function. The indication becomes all the more urgent when there is an organic heart lesion such as valvular disease or coronary artery disease complicating the functional circulatory disturbance caused by the hyperthyroidism. Whereas the heart that is otherwise sound will carry the stress of Graves's disease fairly well for years before becoming insufficient, the already damaged heart gives way. Hyperthyroidism increases the work of the heart in much the same manner as does physical exertion. Just as one would not allow a patient with a crippled heart to engage in frequent or continuous physical exercise, so in hyperthyroidism one must relieve the heart of its additional work as quickly as possible. Only operation will accomplish this expeditiously and thoroughly. In skillful hands the operative mortality is not great. Lahey reports a 3.6 per cent mortality in operations on 138 patients with serious cardiac complications, of whom 110 had congestive heart failure. Of 101 patients who were traced, 95 were returned to full activity, 19 had persistent auricular fibrillation and 6 were still disabled. Other operators have had results equally as good.

Preoperative preparation with compound tincture of iodine is carried out as in all cases of hyperthyroidism. In the presence of auricular fibrillation or heart failure, full doses of digitalis should be given and the fluid intake should be



limited. These patients usually tolerate large doses of digitalis without the appearance of toxic symptoms. Operation should be delayed until the maximum restoration of cardiac function has taken place. It is inadvisable to give quinidine sulfate preoperatively in an endeavor to abolish auricular fibrillation. It is difficult to restore normal sinus rhythm in the presence of active hyperthyroidism. Moreover, even if this result has been achieved, recurrence of auricular fibrillation during or after operation is the rule. It is wiser to try to control the heart rate with digitalis. In patients with auricular fibrillation and no cardiac enlargement or heart failure it is unnecessary to give digitalis, because the auricular fibrillation causes them little circulatory embarrassment. In the majority of patients with auricular fibrillation complicating hyperthyroidism, there is a spontaneous restoration of normal sinus rhythm after subtotal thyroidectomy. If auricular fibrillation persists for two or three weeks following the operation, quinidine therapy should be started unless there is marked cardiac enlargement.

### ADENOMATOUS GOITER

Adenomatous goiters that have existed for decades without symptoms are seen from time to time in patients over 50 years of age. They may, by pressure, cause narrowing of the trachea. Breathing becomes wheezing and even stridulous. Inspiration and expiration are prolonged. Bronchial infection commonly develops. This process leads to increased blood pressure in the pulmonary artery and strain on the right ventricle. Adenomatous goiters may undergo malignant change, or they may give rise to symptoms of hyperthyroidism. In view of the potential troubles that may be caused by these adenomatous goiters, it is wisest to remove them before the

give rise to symptoms and while the operative risk is minimal if any of the aforementioned complications occur operation is no longer a matter of choice, it becomes a necessity, irrespective of the condition of the patient

### MYXEDEMA

Spontaneous myxedema occurs chiefly after middle age but is not common. It is characterized externally by gain in weight, a pasty complexion with puffiness of the face, thickening of the skin and loss of hair. The thyroid gland usually is not palpable. The patient is sluggish, his mental reactions may be dulled, he complains of being cold and does not tolerate exposure to inclement weather. The pulse rate is slow. In long standing cases there is cardiac enlargement and heart failure may develop. The electrocardiogram then shows low voltage of the QRS and T waves. Arteriosclerosis and hypertension are not uncommon accompaniments of myxedema.

Diagnosis rests on the discovery of a basal metabolic rate between minus 35 and minus 40. The blood cholesterol content is characteristically elevated to between 300 and 400 mg per 100 cc. Basal metabolic rates ranging between minus 15 and minus 25 do not justify the diagnosis of myxedema unless there are characteristic symptoms and high blood cholesterol figures. Such lesser depressions of basal metabolism are commonly observed with undernutrition and in thin asthenic persons who complain of a great variety of vague symptoms. They do not indicate a primary diminished thyroid activity. The reduced metabolism is a reflection of the same constitutional disorder that gives rise to the patient's symptoms. Thyroid medication gives such patients little relief.

The symptoms of myxedema can be abolished by admin

istration of desiccated thyroid Dosage must be cautious, and the patient should be kept under close observation during the early stages of therapy Coronary arteriosclerosis and myocardial damage occur so commonly in myxedema that a too hasty whipping up of the metabolism may overtax the heart and quickly induce heart failure, frequent anginal attacks or graver coronary insufficiency It is well to start with 1 gr of desiccated thyroid once a day This should be continued for one or two weeks If there are no untoward symptoms, the dose may be doubled for the next week or two In the course of the following weeks further increases in dosage up to a maximum of 5 gr a day are permissible The maintenance dose is the smallest dose that will maintain a normal metabolic rate and give relief from symptoms If cardiac symptoms arise during the course of medication with desiccated thyroid, the drug should be stopped until the symptoms subside It should be recommenced with half the previous dosage, and the patient should be watched closely At times, prolonged treatment with small doses of desiccated thyroid eventually relieves the myxedematous state and restores the metabolism to normal More often, cardiac symptoms arise when the basal metabolic rate reaches a level of from minus 20 to minus 25 In such cases it may be impossible and inadvisable to attempt completely to abolish the myxedematous state Just enough desiccated thyroid should be given to relieve the patient of the gravest symptoms Only by such measures can serious cardiac complications be avoided

### CARCINOMA OF THE THYROID

Carcinoma of the thyroid rarely develops in diffuse toxic goiters It is encountered in 4 to 7 per cent of nodular or adenomatous glands and is especially prone to develop in a

solitary adenoma Like other thyroid disorders, it is more common in women, 80 per cent of patients being female It occurs at all ages from childhood on, but the average age is 50 years An enlarged thyroid or a mass in the gland antedates the development of carcinoma by seven years, on the average The two common forms are adenocarcinoma, in which the tumor growth takes the form of the thyroid follicles which may contain colloid, even in distant metastases or of a papillary adenocarcinoma The growth may be locally invasive, break through the capsule of the gland, invade the veins and by pressure on the trachea and esophagus cause dyspnea and dysphagia There is local extension to the cervical lymph nodes, Metastasis through the blood stream, which may occur when the primary lesion is still minimal in size, localizes most commonly in the lungs and in the bones

Diagnosis is difficult, and 80 per cent of cancers of the thyroid are first recognized by pathological examination of a gland that has been removed for other reasons A recently growing struma that is very hard and that is fixed to the surrounding tissues and does not ascend in the neck when the patient swallows should suggest malignancy The basal metabolic rate is usually normal Hyperthyroidism is not seen with malignant tumors of the thyroid gland Hypothyroidism in a case suspected of malignancy suggests that the lesion is due rather to chronic thyroiditis so called Riedel's struma

Once the diagnosis has been made, operative removal is indicated, even if a few metastases are demonstrable in the lungs or bones Operation should be followed by radiotherapy to the neck and to the metastases If operation is impossible, radiotherapy should be carried out It is effective chiefly in the case of papillary tumors

Five year survivals are reported in 80 per cent of cases in which the diagnosis was first made by pathological study of

*THE PATIENT PAST FIFTY*

the gland, in 40 per cent of those first diagnosed at operation and in 20 per cent of those diagnosed before operation. These figures indicate the advisability of removing all nodular goiters without too much delay.

## Chapter Eighteen

# DISORDERS OF THE BLOOD

THERE are no distinctive changes in the blood picture that come with aging. Persons over the age of 60 have somewhat lower values for hemoglobin and red blood cells than do those of younger ages. As is the case throughout life, the figures for women are lower than those for men. There is no characteristic alteration of the number and relative proportions of the white blood cells with advancing years. The total blood volume and plasma volume tend to diminish with advancing age, and more so in men than in women.<sup>1</sup>

## SECONDARY ANEMIA

Secondary anemia is due to iron deficiency in the body. This may result from massive or slow insidious loss of blood or from deficient ingestion or absorption of iron. Old people often exhibit idiosyncrasies of diet and may subsist largely on starchy foods and milk which contain very little iron. Lack of teeth or digestive disturbances that interfere with digestion or absorption of food may contribute to limitation of food intake. In time an iron deficiency results, manifested by a secondary anemia. It is a common accompaniment of many

<sup>1</sup>Gibson, J. C. and Evans, W. A. Jr. "Clinical Studies of Blood Volume," *J. Clin. Investigation* 16:317, 1937.

chronic diseases. Massive hemorrhage in older persons results most often from nosebleed in the presence of essential hypertension or from hemorrhage from a peptic ulcer. Slow repeated or persistent hemorrhage is associated most commonly with bleeding hemorrhoids. The anemia observed in patients with diaphragmatic hernia is caused by weeping of blood from the congested imprisoned gastric mucosa. Gastric achlorhydria which is so common in the aged may contribute to the development of anemia by making more difficult the absorption of iron.

In every case of secondary anemia the physician should first try to ascertain its cause and in particular rule out the presence of a neoplasm especially of the gastrointestinal tract and chronic nephritis with kidney insufficiency. Estimation of the sedimentation rate of the red blood cells is a useful aid in ruling out malignancy. The symptoms of secondary anemia depend on its degree. In the milder cases the patient complains of weakness and because so commonly the cause is dietary he may have other symptoms of dietary insufficiencies. If the hemoglobin drops below 60 per cent symptoms of dyspnea, palpitation, ready fatigue and possibly edema appear. If there is an accompanying heart ailment with reduction of cardiac reserve these signs become more prominent.

The diagnosis rests on examination of the blood which reveals lowering of both hemoglobin and red cells with a low color index. The red cells are small or of normal size. The color of the skin of older patients is very deceptive and often a patient whose blood count is normal appears to be anemic. Treatment involves control of any bleeding that may exist, the provision of an adequate well balanced diet and the administration of iron. This is best given in the form of ferrous sulfate or carbonate 5 gr. three times a day.

Ingestion of iron often induces constipation so that it may be necessary to give cathartics to keep the bowels open. Liver extract is of no benefit in secondary anemia. If the patient responds poorly to iron one or more transfusions may be needed.

### HYPERCHROMIC (PERNICIOUS) ANEMIA

Pernicious anemia is encountered chiefly between the ages of 40 and 70. About 60 per cent of cases occur after age 50 and 30 per cent after age 60. The maximum number of cases are between the ages of 50 and 60. Since pernicious anemia is caused by the absence of the intrinsic factor secreted by the stomach and since the loss of such a secretion is at least as the loss of hydrochloric acid may be expected to become more frequent with the years the age incidence is quite understandable. When one allows for the lesser number of persons living at the higher age groups it is evident that the actual age specific incidence of pernicious anemia increases with the years.

The symptomatology of pernicious anemia in the elderly does not differ from the usual picture described in the text books of medicine and it need not be elaborated in detail in this volume. Characteristic are the hyperchromic macrocytic anemia with a high color index and accompanying leukopenia, the elevated icterus index, the absence of free hydrochloric acid in the stomach even after the injection of histamine, the sore tongue with atrophied mucous membrane, numbness and tingling of the extremities with signs of combined sclerosis of the spinal cord. Loss of vibratory sense when tested with the tuning fork is the earliest neurological sign.

Certain special problems arise with the diagnosis of per-



pernicious anemia in older persons. The clinical picture may simulate cancer of the stomach. As a rule the blood picture showing a macrocytic instead of a microcytic anemia leads to the correct diagnosis. Patients with cancer of the stomach and marked anemia usually have lost much weight, an abdominal mass may be palpable and there is blood in the stool. The icterus index is not elevated. The sedimentation rate of the red blood cells is of no aid in diagnosis. I have seen a number of patients with pernicious anemia in whom the sedimentation rate was very rapid, and became normal when a remission was induced. In every case of unexplained anemia, roentgen examination of the stomach should be undertaken. The occurrence of pernicious anemia and carcinoma of the stomach in the same patient is so frequent as to suggest some basic relationship between the two diseases. Autopsy studies show an incidence of 12.3 per cent of gastric cancer in patients with pernicious anemia. Rigler and his associates<sup>2</sup> in routine roentgen studies of 211 patients with pernicious anemia discovered 8 per cent with carcinomas and 7 per cent with benign polyps of the stomach. Such polyps are often precursors of carcinoma. The coincidence is so great that it would seem wise to subject patients with pernicious anemia to annual roentgen studies of their stomachs and to be on the watch for digestive disturbances that may signalize the development of a gastric neoplasm. Cirrhosis of the liver may give rise to a hyperchromic macrocytic anemia that may be confused with pernicious anemia. In these cases the cirrhosis is apt to be far advanced. The history of the case, marked splenomegaly, possibly ascites and the discovery of esophageal varices serve to differentiate the two conditions.

Older patients commonly have coronary arterial disease,

<sup>2</sup> Rigler, L. G., et al. "Pernicious Anemia and the Early Diagnosis of Tumors of the Stomach," J. A. M. A., 128:426, June 9, 1915.

and the impaired heart muscle does not stand up under the severe prolonged anemia. Consequently, angina pectoris may be a leading symptom. With further myocardial damage congestive heart failure may supervene. This picture was very common in the days before liver therapy was introduced but is rare today. The cardiac symptoms yield when the anemia is corrected.

*Treatment*—This centers on giving enough liver extract to restore the blood count to normal and to maintain it there. During the anemic stage it is best to give the liver extract intramuscularly, at first 15 U S P units every two or three days. When the hemoglobin and red blood cell count approach normal, the intervals between injections can be lengthened to a week. The maintenance dose varies with different patients, 15 units every one, two or three weeks is usually required. If preferred oral preparations can be given daily instead. In the presence of neurological symptoms, treatment should be particularly intensive and there must be no relaxation when a remission has set in.

### POLYCYTHEMIA VERA

True polycythemia occurs chiefly after age 50. Its cause is unknown. It is characterized by a great increase in the hemoglobin in the blood and in the number of red cells. The hemoglobin figures may reach 125 to 150 per cent and the red cells may number from 7,000,000 to 10,000,000. The blood volume is increased. This serves to distinguish it from secondary polycythemia in which the volume of blood is unaltered. The number of white blood cells, too, is elevated to between 15,000 and 20,000 cells, and there is a relative increase in the polymorphonuclear leukocytes.

The disease comes on insidiously and the patient complains

chiefly of cerebral symptoms—headache, or fullness of the head, vertigo, lassitude and irritability. He appears plethoric and of a dusky red color, particularly in the face. As a rule the spleen and liver are enlarged. Hemorrhages often occur. Thus there may be epistaxis, menorrhagia or even massive intestinal bleeding. I have seen a patient in whom the bleeding suggested a peptic ulcer. The roentgen picture of the chest reveals increased vascular markings of the lungs and, at times, curious discrete, sharply outlined circular shadows that may represent thrombosed pulmonary veins. The course of the disease runs from 5 to 10 years. It is characterized by a great tendency to venous and arterial thromboses. *Thrombosis may occur in any vein or artery.* The most common sites are in the cerebral and coronary arteries and the arteries of the legs. Coronary thrombosis and cerebral vascular accidents are the usual causes of disability and death. At times after many years the blood picture changes to that of aplastic anemia, or of leukemia, and the patient dies from anemia and exhaustion of his bone marrow.

The best treatment is repeated phlebotomy, performed with sufficient frequency to maintain the blood count at approximately normal figures. Acetylphenylhydrazine, a hemolytic poison, reduces the red blood cell count but is little used today because of the danger of excess hemolysis. Radiotherapy to the long bones and spleen is often beneficial. The diet should be poor in iron.

Secondary polycythemia is encountered most often with advanced pulmonary emphysema. In this condition, too, phlebotomy is helpful.

### LEUKEMIA

There is some variance in the figures of the frequency of the leukemias at different ages. It is usually held that the

acute leukemias, as well as chronic myelogenous leukemia, are infrequent in older individuals. All authors agree that chronic lymphatic leukemia occurs chiefly after age 50. In a series of 143 cases,<sup>3</sup> however, one half of the subjects with both the acute and chronic myelogenous leukemias were over 50 and 40 per cent of those with acute lymphatic leukemia and 88 per cent of those with chronic lymphatic leukemia were over 50. Monocytic leukemia, too, is most common after the fifth decade. All forms of leukemia, but particularly the chronic lymphatic form, are more common in males than in females.

Leukemias in elderly persons do not differ from those in the young. Anemia, loss of weight, enlargement of the lymph nodes, liver and spleen are the characteristic features of the chronic forms of the disorder. The basal metabolic rate is elevated. The prognosis for all leukemias is poor. Patients with chronic forms, particularly lymphatic leukemia, may survive many years, although the average duration is from three to four years. Treatment is unsatisfactory. Fowler's solution, given to the point of tolerance, seems to benefit some patients with chronic myelogenous leukemia. Isaacs recommends the employment of radiotherapy only when there is progressive anemia or when there are marked nervousness, weight loss, tachycardia, sweating and sleeplessness. It may be useful in reducing the size of a very large and heavy spleen. Repeated transfusions are of benefit when there is anemia.

#### ACQUIRED HEMOLYTIC JAUNDICE

Hemolytic jaundice may have its onset after the age of

<sup>3</sup> Kirshbaum J D and Preuss F S "Leukemia" Arch Int Med, 71 777, June 1943

50, it has been observed in the eighth decade <sup>4</sup> In such cases there is no family history or previous indication of the disorder There is rapid development of hemolytic anemia, with weakness palpitation dyspnea, pallor and jaundice The spleen and liver are enlarged In some cases there is increased fragility of the red blood cells in hypotonic salt solutions together with spherocytosis Splenectomy may lead to improvement and may, indeed be life saving It is particularly effective in patients with increased erythrocyte fragility

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<sup>4</sup>James D F and Evans L R "Splenectomy for Acquired Hemolytic Jaundice in the Aged" *New England J Med* 233 143 Aug 2 1945

## Chapter Nineteen

# DISEASES OF THE EYES

THE EYES manifest many signs of aging. The orbital fat becomes absorbed so that the eye sinks deep into its socket. The eyelids lose their tonicity, so that eventually the upper lid may droop and the lower lid may be everted. The sclera takes on a yellowish color as a result of fatty changes. The color of the iris fades, and the pupil becomes small and less mobile. The arcus senilis, the circular lipid infiltration at the circumference of the cornea, may occur among younger persons, but becomes increasingly frequent with advancing years. Its incidence at different ages is shown in Table 7<sup>1</sup>.

TABLE 7 —INCIDENCE OF ARCUS SENILIS

AGE	ARCUS SENILIS %	
	MALE	FEMALE
30-39	5.5	0
40-49	10.0	4.0
50-59	26.0	18.0
60-69	37.0	30.0
70-79	73.0	17.0

With age comes loss of visual acuity. This is shown in all of the physiological functions of the eye, the visual fields become smaller and the power of accommodation is lessened. Old people require greater intensity of light for close vision.

<sup>1</sup>Boas, E. P. "Arcus Senilis and Arteriosclerosis," J. Mt. Sinai Hosp., 12:79 May-June 1945.

and thus should have stronger illumination for reading or other similar use of their eyes. They see less well in the dark. The steady decline in the capacity for dark adaptation that comes with aging is due to the progressive decrease in the size of the pupil.<sup>2</sup> There is a lessened ability to match colors after age 65.<sup>3</sup> The lens grows less and less elastic and calls for ever greater accommodative efforts on the part of the patient. When such accommodation can no longer be made, the condition of presbyopia develops, the patient cannot focus on nearby objects, cannot read and needs glasses for near vision. He has no difficulty with far vision. Bernstein has demonstrated a definite correlation between the age of onset of presbyopia and longevity. On the average, those who develop presbyopia early die at younger ages than those in whom presbyopia develops late in life, and these early presbyopes tend to die of arteriosclerotic heart disease.<sup>4</sup> The so called second sight, a condition in which a person who has required glasses for near vision can do without them in late life, is due to an increasing shallowness of the anterior chamber of the eye and increased refractive power of the nucleus of the lens.

Blepharitis and conjunctivitis are common in older persons. The palpebral conjunctiva becomes congested and red. Cilcareous concretions consisting of dead epithelial cells and hardened mucus may form in the serous glands of the conjunctiva.

Some of these senile changes in the eye require treatment.

<sup>2</sup> Robertson G. W. and Yuckim J. "Effect of Age upon Dark Adaptation," *J. Physiol.* 103:1 June, 1944.

<sup>3</sup> Smith H. C. "Age Differences in Color Discrimination," *J. Gen. Psychol.* 29:191 1913.

<sup>4</sup> Bernstein F. "Law of Physiologic Aging as Derived from Long Range Data on Refraction of the Human Eye," *Arch. Ophthalm.* 34:378, Nov-Dec, 1945.

When the upper lid droops enough to interfere with vision and when eversion of the lower lid leads to tearing and conjunctivitis, these deformities can be corrected by simple plastic operations. Calcareous concretions of the conjunctiva should be removed. Presbyopia calls for the prescription of glasses for near vision. The strength of the glasses should be determined not alone by the degree of impairment of accommodation but also by the chief occupation of the patient. Thus, a person who reads and writes a good deal should have an adjustment that will allow for nearer vision than a musician, an artist or a typist. At times it is well to provide an intermediate pair of glasses that afford easier vision at card playing or at some other activity that calls for less close application.

Disease conditions that lead to failing vision of the aging person are primarily vascular changes of the fundus: central macular senile degeneration, cataract and simple noninflammatory glaucoma.

Vascular changes of the fundus characteristic of the aged are due to simple retinal arteriosclerosis and are unrelated to arterial hypertension. The arteries of the eye grounds become tortuous, and there may be degenerative lesions in the retina. Hemorrhages occur but are infrequent. The more striking picture of narrowed arteries, edema, exudates, hemorrhages and scars are associated with arterial hypertension and do not differ from similar vascular lesions at younger ages. Senile degeneration of the macula is probably also of vascular origin. The condition is bilateral. Central vision becomes impaired, and when the process is complete there is a central blind spot. Treatment is of no avail.

Cataract is the chief cause of blindness after the age of 65. Although cataract formation is closely related to aging of the lens, hereditary factors play a rôle in some cases. Cataracts



may appear in siblings when they reach the same age. Nuclear changes with some haziness of the lens and minor opacities may be found in over 90 per cent of persons past the age of 65. Many of these small opacities do not progress to full cataract formation. The later in life that opacities of the lens appear, the less likely are they to cause loss of vision. A cataract that begins after the age of 60 is far less apt to mature than one that begins one or two decades earlier. If cataract occurs in only one eye, the patient may be unaware of it, for the good eye takes on the task of both. But gradually there develops some interference with vision. When an early cataract is centrally placed, the patient's near vision may improve for a time, because of the increased refractive power of the denser lens. But distant vision is impaired, and bright objects may appear blurred or double. With a central opacity, the patient sees better in dull light, which allows the pupil to dilate, permitting light to pass through the peripheral parts of the lens. In the case of peripheral opacities, vision is better in a bright light, for the central part of the lens is clear and the contracted pupil shuts out the distorted images that arise by light passing through the peripheral opacities.

The thought of cataract with possible resultant blindness strikes terror into the hearts of most persons. Therefore when a cataract is discovered the physician should take great pains to explain the full situation to the patient. He must make clear that most cataracts mature very slowly, that for years the patient may preserve satisfactory vision and that if blindness finally comes on it can usually be relieved by operation. The patient's vision can be aided in various ways. In the case of central cataract, if there is no glaucoma, the instillation in each eye every morning of a drop of 1 per cent homatropin and 5 per cent ephedrine solution will dilate the pupil sufficiently to improve vision. Reading is facilitated by the

use of a rubber sheet, about half the size of the page of a book. This contains a slit  $\frac{1}{4}$  in long and  $\frac{1}{8}$  in wide. It is placed on the page of reading matter so that one line of type shows through the slit. This device makes reading easier because it lessens the amount of light reflected from the white page and so lessens pupillary contraction and eliminates the blurred images of peripheral vision. When the cataract finally becomes mature it should be extracted. Before operation, the surgeon should make sure that retinal function is still intact. The pupil should react to light, and the patient should perceive light.

The characteristic glaucoma of the aged is simple non-inflammatory glaucoma. It has a familial occurrence, gives rise to little pain, pursues an insidious course and may result in serious permanent impairment of vision before it is recognized. The patient complains of occasional slight blurring of vision, commonly more marked in one eye. When in a dim light or when emotionally upset he may experience some pain in the eyes, after visiting a motion picture performance his eyes may feel strained or uncomfortable. There is some impairment of vision and the patient has his glasses changed frequently without relief. These symptoms progress very slowly. Only when the condition is far advanced does the patient notice halos or rainbow effects about lights. Examination may reveal few physical signs. The intra ocular tension is not continuously or greatly elevated; it is apt to be higher in the late afternoon or when the patient is under strain. The hardness of the eyeballs should be gaged by palpation with the fingers, and careful measurements with the tonometer should be made. The pupils may be unequal and may react sluggishly to light. Central vision for reading is at first preserved, but careful examination reveals subnormal vision in one or both eyes, with irregular contraction of the peripheral

field of vision. Cupping of the optic disk, observed through the ophthalmoscope, is a late phenomenon. All too often the diagnosis is overlooked until it is too late, and complete blindness results. This error is especially common when cataract and glaucoma occur simultaneously. The failing vision is ascribed to the cataract, and the glaucoma is allowed to progress. Finally, when the cataract is ripe, operation does not restore the vision that has been completely destroyed by the glaucoma. Aging persons should have their eyes checked yearly, and every examination must include measurement of the pressure of the eyeball. The treatment of glaucoma should be in the hands of a skilled ophthalmologist. He will determine whether conservative treatment with miotics or operation is indicated.

Acute glaucoma occurs chiefly between the ages of 45 and 65. It has no distinctive characteristics when it appears in aging persons.

## DISEASES OF THE SKIN

THE SKIN of the aging person loses its elasticity and becomes wrinkled. The epidermis, especially the malpighian layer, becomes thin, the basal layer is pigmented, the papillae are flattened, and the elastic fibers and the subcutaneous fat disappear. Gradual atrophy of the sweat and sebaceous glands leads to dryness and roughness of texture. Eventually when the skin grows very thin it becomes smooth and glossy. Seborrheic keratoses appear, particularly on the trunk and face. They are slightly elevated, greasy, flat, yellowish-brown to black plaques and may be numerous. They have no clinical significance and can be ignored. Excessive bathing and the use of strong soaps remove the scanty fat supply of the aging skin, increase its dryness and lessen its resistance to external irritants. An emollient such as cocoa butter or an ointment made of equal parts of lanolin and petrolatum often gives relief when the skin is very dry.

Graying of the hair and baldness are accepted by the laity as evidences of aging. Yet there is great variability in the age at which these phenomena appear. Hereditary influences play a strong rôle. The average age at which gray hairs first appear is  $34.2 \pm 8.6$  years for white men and  $43.9 \pm 10.3$  years for Negro men. In general, however, light hair becomes gray later in life than does dark hair. While the hair of the

scalp thins, the hair of the nose, ears and eyebrows increases in amount and becomes thick and bristle-like. In women, hair appears on the upper lip and on the chin.

Senile keratoses are small, elevated horny growths covered with scales that appear on the exposed parts of the skin. These keratoses may undergo carcinomatous metaplasia—particularly if they occur at a site that is constantly irritated by clothing. Such growths should be removed, preferably by radiation therapy. They tend to recur and often new ones form.

Leukoplakia of the mouth occurs as white thickened patches on the mucous membrane of the cheeks or of the tongue. It may result from syphilis, from excessive smoking or chewing of tobacco or from imperfect teeth. Leukoplakia commonly forms the nidus for the development of cancer. Pipe smokers may develop cancer at the tip of the tongue. Those who chew tobacco are apt to develop a lesion on the inner aspect of the cheek where the quid is held. Ragged stumps of teeth or poorly aligned bites may produce sufficient irritation of the mucous membrane to induce leukoplakia or cancer. In the presence of leukoplakia, smoking must be stopped completely. The teeth should be put in good condition. In old people, the physician should pay strict attention to the hygiene of the mouth, for neglect on the part of the patient is common.

Basal cell epithelioma, or rodent ulcer, is not confined to the aged. The average age at onset is given as 45 years. It is a slow growing lesion that begins most often on the face above the level of the nostrils as a warty growth that ulcerates. It remains superficial and for a long time does not penetrate deeper than the superficial fascia. Gradually the growth extends at the periphery, and the center ulcerates and is filled with granulation tissue. The edges of the ulcer are raised and

*nodular* Ultimately it may invade and erode deeper tissues and neglected cases are seen in which the eye ear or nose has been destroyed or the maxillary sinus invaded. The lymph nodes are never involved. Early diagnosis is essential, and every suspicious lesion must be subjected to biopsy. With modern methods of radiotherapy, fully 98 per cent of these growths can be cured by the use of appropriate techniques.

Squamous cell epithelioma has its highest incidence in the seventh decade and usually arises from senile keratoses. It is a true malignant neoplasm which grows rapidly and metastasizes. The base of the lesion is infiltrated, ulceration and bleeding are the rule. Every such lesion of the skin should be regarded with suspicion, and a section removed for biopsy whenever there is the slightest doubt. Early epitheliomas can usually be cured by radium or roentgen therapy, but treatment must be individualized. In some cases excision or electrocautery and in others curetting and the application of caustics followed by radiotherapy are the methods of choice.

changes. It is always essential to rule out such causes as pediculosis, scabies, eczema, diabetes, Hodgkin's disease and azotemia. When no definite cause can be determined, when the skin is dry, thin and atrophic, when the condition is aggravated by cold and by frequent bathing, one may assume that one is dealing with true senile pruritus. Treatment in the pa... -2 soothing emolli... addition of son... treatment with the sex hormones may give complete relief from senile pruritus. Women are given injections of 1 mg. estradiol di propionate and men 25 mg. of testosterone propionate about

every five days until the symptoms are controlled. If the injections are stopped the symptoms will return in a few weeks so that treatment must be continued indefinitely but the injections can be spaced at longer intervals. Some patients require higher dosage some less. In each case the optimum dose must be determined by actual clinical trial.

### VARICOSE VEINS AND ULCERS OF THE LEGS

Varicose veins and their sequelae may first appear in youth. They occur most frequently in early middle life. If untreated they cause increasing disability and in older patients they are often responsible for chronic eczema, infection and ulceration of the legs. Varicose dilatation of the leg veins may be a primary condition or it may represent a collateral circulation secondary to phlebitis and occlusion of the deep veins of the leg. The cause of the development of primary varicosities is not understood. There is a marked hereditary factor for a familial incidence of varicose veins is found in over one half of all cases. Absence or incompetence of the valves in the perforating veins of the sphenous systems probably represents the underlying defect. Occupations that compel standing for many hours, pregnancy and abdominal tumors may contribute to the venous stasis and accelerate the process.

In well marked cases the internal sphenous veins stand out as thick cords extending down the thighs. In the popliteal space and in the leg are large bunches of tortuous dilated veins. In milder cases only small superficial veins are dilated as spider bursts or only a few of the veins of the legs are dilated. When the varicosities are secondary to occlusion of the deep veins anastomotic channels to dilated veins on the abdomen and trunk are usually visible.

The patient complains of heaviness. In time, edema of the legs appears. The and subcutaneous tissues suffers as a result of impairment of blood flow, and the skin becomes and indurated. As a result of repeated attacks

the skin is common and is often made worse by infection of the feet. Eczematous eruptions commonly occur. Finally, the tissues break down, and ulcers of the lower extremities develop. These ulcers are very persistent and heal with difficulty.

**Treatment**—When there is much eczema and this must be brought under control before the ulcers are treated. The patient is put to bed and the ulcers are dressed. If there is extensive weeping eczema wet dressings are applied. Five per cent alcoholic solution of gentian violet is applied to the ulcers and to areas infected by fungus. When the infection subsides and the skin becomes dry, the ulcers are dressed with a dry dressing.

particularly when the internal saphenous vein is utilized. The injection of the veins with a sclerosing solution is a satisfactory results. This is a surgical procedure. A 1 per cent rhinate in a 5 or 10 per cent solution is favored by some operators. Occasionally allergic reactions to this solution are countered. Other operators use quinine hydrochloride and urethane. If there are marked incompetence of the femoral valve and dilatation of the femoral vein, injection alone will not be successful. The ligation of this vein at the saphenofemoral junction is preferred out, after first establishing the competence of the



veins The superficial veins are then injected Treatment carefully carried out is successful in the majority of cases There may be recurrences due to recanalization of the induced thrombi or to marked incompetence of some of the communicating veins between the deep and superficial venous systems At times ulcers do not heal and have to be excised, the exposed area being covered by skin grafts Varicose veins that arise as compensatory channels as a result of thrombosis of the deep veins of the leg should not be obliterated If they cause distress, the patient should wear bandages or elastic stockings

## Chapter Twenty One

# DISEASES OF THE NERVOUS SYSTEM

IN ADVANCED age the brain undergoes involution. There has been much controversy, just as there has been in the field of arteriosclerosis, as to whether these changes represent the aging process as such or whether they are the result of the innumerable vascular and toxic insults to which the brain has been subject during the long years of life.<sup>1</sup> There is great variability in the occurrence of senile changes in the brain. Anatomic study of the brains of some persons who have died in the ninth decade has revealed remarkably few changes, these were individuals who had retained their full mental faculties up to their death. In general the aged brain is shrunken and firm, its surface is wrinkled, its fissures wide, its ventricles dilated. The process is especially marked in the frontal lobes. The gray matter of the cortex may have a worm eaten appearance. Many of the nerve cells are atrophied, and there is an increase of intracellular pigment. The nerve fibers are diminished in numbers. There is an increase in the corpora amylacea. There may be many small foci of softening. The so called senile plaques, dense circular areas with an amorphous center, especially studied by Alzheimer and by Simehowsky, are interpreted by many as characteristic

<sup>1</sup> Gilney, F. "Aging of the Human Brain," Bull. New York Acad. Med., 4: 1125, 1928.

of senile involution of the brain. In normal persons they do not occur before the age of 75 or 80 and then are few in number. After the ninetieth year they are always found. In persons who die at earlier ages with senile dementia these plaques are present in great numbers and they are particularly numerous in precocious senile dementia (Alzheimer's disease).

Old age brings with it a general dulling of the senses. Vision becomes dim, hearing less acute, the senses of taste and smell less discerning. The taste buds in the circumvallate papillae become fewer with the years, especially after age 75.<sup>2</sup> The number of fibers in the olfactory nerves decreases progressively throughout life.<sup>3</sup> Responses to pain and to touch are less marked. With every decade there is a slight diminution in the vibratory sensibility of the lower extremities. This becomes marked after age 50 and in many older persons vibratory sensibility of the legs is absent. This change is believed to be due to vascular lesions in the posterior columns of the spinal cord.<sup>4</sup> Motor disturbances reflect lesions of the extrapyramidal tracts. The body tends to assume attitudes of flexion.<sup>5</sup> The head and neck are bent forward and the elbows, wrists, hips and knees assume the position of semi-flexion. Rigidity of the limbs may develop. There is a diminution of muscular movement, what may be called a poverty and slowness of movement, with a tendency to prolonged immobility. The senile gait, with the legs apart, giving a widened base to the body, the body and legs some

<sup>2</sup> Arey, L. B. et al. "The Numerical and Topographical Relations of Taste Buds to Human Circumvallate Papillae throughout the Life Span." *Anat. Rec.* 64:9, 1935.

<sup>3</sup> Smith, C. G. "Age Incidence of Atrophy of Olfactory Nerves in Man." *J. Comp. Neurol.* 77:589, 1942.

<sup>4</sup> Pearson, G. H. J. "Effect of Age on Vibratory Sensibility." *Arch. Neurol. & Psychiat.* 20:482, 1928.

<sup>5</sup> Critchley, M. "Neurology of Old Age." *Lancet* 1:1119, May 31, 1971.

what flexed and the steps shortened is the direct result of these extrapyramidal changes. When these changes are sufficiently advanced they may lead to a propulsive gait. When well developed all of these phenomena give the typical picture of parkinsonism.

Tremors are common. They are rapid and fine and involve the head, the lower jaw, the hands and the forearms. They are exaggerated by emotion and fatigue and abolished during sleep. Simple senile tremor is not associated with rigidity. The reflexes may be diminished and the knee and ankle jerks are often absent.

The most common neurological disorders of the aged are caused by arteriosclerosis of the cerebral arteries. These have been discussed under the section on arteriosclerosis. They may manifest themselves as frank hemiplegias or give the clinical picture of multiple cerebral vascular insults or of cerebral softening. Cerebral softening due to gradual atherosclerotic and thrombotic occlusions of cerebral arteries is the typical lesion of the aged. There may be prodromata such as headache, vertigo or somnolence followed in a few days by paralysis of one side of the body or of one limb. There may be mental confusion or impairment of memory. Characteristically there is a slow progression of symptoms over a period of years. There are more and more loss of motor function and progressive destruction of the intellectual faculties. Eventually loss of sphincter control develops and the classic picture of arteriosclerotic dementia sets in. Multiple bilateral cerebral lesions may lead to the syndrome of pseudobulbar palsy. The facies are expressionless but the patient has paroxysms of laughing and crying without cause. The mouth is held partly open and there is drooling of saliva. The voice is nasal without timbre and without expression. The motor signs are those of multiple lesions of the pyramidal tract.

## PARALYSIS AGITANS OR PARKINSON'S DISEASE

Paralysis agitans is a disease of later life characterized by tremor and muscular rigidity. It should be distinguished from the Parkinson syndrome that follows epidemic encephalitis and occurs in young persons. The lesions responsible for this disorder are widespread in the cortex, the corticothalamic connections and in the basal ganglions. The substantia nigra is most constantly involved, and the pyramidal system is spared.<sup>6</sup> Some cases are the end result of an unrecognized encephalitis, others of arteriosclerotic softening, and in still others the cause cannot be determined.

The average age at onset is between 50 and 55. Progress of the disease is slow. It may last 10 to 30 years and does not appreciably shorten the life of its victim. The disease begins insidiously, often with rheumatoid pains and weakness of the extremities. Tremor is the first distinguishing sign. As a rule it first becomes manifest in the small muscles of the hands and involves the thumb and index finger, which undergo from four to seven flexions a second, giving rise to the so called pill rolling movement. Gradually in the course of two to four years the affected area extends to involve the wrist and the forearm. The muscles of the calf and the toes may also give evidence of the disturbance. Finally the muscles of the face become affected, there is tremor of the tongue and of the lips and there may be a tremor of the head. The facial muscles themselves are usually immobile and rigid and give rise to the expressionless, masklike facies. The tremor is present at rest, becomes less on voluntary motion and ceases during sleep. At times the symptoms are restricted to one half of the body.

<sup>6</sup> Benda, C. E. and Cobb, S. "On the Pathogenesis of Paralysis Agitans." *Medicine* 21:95 May 1942.

Muscular rigidity is an essential part of the clinical picture. This tends to keep the trunk and extremities in the position of flexion. The head is flexed on the trunk, the trunk is bent forward and the arms and legs, even in walking, are held in the position of semiflexion. The muscular rigidity offers resistance to passive stretching of the muscles, and it is difficult to straighten the patient's limbs. There is great delay in initiating movement, and a considerable latent period elapses between the giving of an order and the execution of a movement on the part of the patient. Movement is slow, deliberate and difficult. For the same reason speech is slow, deliberate, low and monotonous. Walking is seriously affected. The patient gets up with difficulty and starts off with a slow shuffling gait. Then bent forward, he takes faster and faster small steps, exhibiting the typical propulsive gait. In extreme cases he will fall forward if his progress is not arrested. While walking, the body is rigid, and there are no automatic and associated movements, such as swinging of the arms. The body moves as though made of one piece—like a piece of machinery to use Parkinson's simile. As a result of the muscular rigidity and the difficulty in executing movements, the patient tires very easily.

Throughout the course of the disease the mind remains clear, even though the lifeless facies, the slow reactivity and the halting speech often suggest mental impairment. For years there may be only slight tremor of a few fingers, but gradually and inevitably more and more muscle groups become involved and greater and greater rigidity sets in. Eventually the patient becomes completely helpless as a result of the generalized rigidity. He can no longer walk, cannot feed himself or execute the simplest movements to help himself. This state may last for five or six years before death finally brings release.

*Treatment*—General treatment is concerned with maintaining nutrition and assisting the patient to keep up and about and active. Massage, warm baths and passive movements of the extremities are helpful. For years belladonna and scopolamine have been used with varying results. More recently, exceptional virtues have been attributed to Bulgarian belladonna. Further studies have revealed that its efficacy lies not in the root itself but in the method of extraction. A vinous extract of the American roots is as efficacious as the extract of the Bulgarian variety. Apparently extraction with white wine gives a different preparation than extraction with alcohol. Such a preparation is available under the name of Vinobel. Each tablet contains 0.4 mg. of the total alkaloids. Dosage is commenced with 1 tablet a day and the amount is increased by 1 tablet daily until the optimum effects are obtained or until toxic symptoms appear. The average dose required for each patient differs, on the average, 7 to 10 tablets a day are needed. Some patients do well on 3 tablets others may require 20 or more. Toxic symptoms are those of atropine poisoning: mydriasis, loss of accommodation, dryness of the mouth, a hot flushed skin and tachycardia. Symptomatic improvement of parkinsonism is manifested chiefly by lessening of the rigidity and salivation and increase in strength and sense of well being. The tremor is little affected. The postencephalitic type of parkinsonism responds especially well to this treatment, and some authors claim maximum improvement in over half the cases. The same results can be obtained by the use of atropine sulfate. The patient is given a  $\frac{1}{2}$  per cent solution of which each drop contains  $\frac{1}{200}$  gr. of atropine. The initial dosage of 1 drop three times

<sup>1</sup> Fahrig, H. D. and Zeligs, M. A. "Treatment of the Postencephalic Parkinsonian Syndrome with Desiccated White Wine Extract of U.S.P. Belladonna Root." J. A. M. A., 117:332 Aug. 2, 1941.

a day is gradually increased to between 5 and 10 drops three times a day. Older persons with paralysis agitans due to degenerative disease benefit less often and may not tolerate the necessary large doses of the drug. They should, however, be given a trial, because some are helped. In the presence of glaucoma, belladonna products should not be used, and when there is accompanying cardiovascular disease they should be employed with care.

Highly satisfactory results have been reported from the administration of vitamin B<sub>6</sub> (pyroxidine hydrochloride)<sup>8</sup>. The patients receive from 50 to 100 mg. of pyroxidine hydrochloride intravenously daily for from two to four weeks. The maintenance dose must be adjusted to the needs of the patient. Astonishing results have been reported, with marked reduction in rigidity and tremor and great gain in strength. Even long standing, advanced cases have been improved. The arteriosclerotic type of the disease responds better than the postencephalitic form.

The recently isolated active principle of curare, tubocurarine, is being employed to relieve muscle spasms of all sorts. Tubocurarine depresses the myoneural junction. Dosage can be adjusted to block abnormal impulses without loss of voluntary power of the muscle. Given to patients with parkinsonism, it relieves rigidity but not the tremor.<sup>9</sup> Patients get great relief from such relaxation, often lose the pain from muscle tension and are able to sleep. A 3 per cent solution of tubocurarine is prepared in 48 per cent wax in peanut oil. This preparation can be sterilized. The average dose is 1 cc. subcutaneously or intramuscularly. The action

<sup>8</sup> Baker, A. B. 'Treatment of Paralysis Agitans with Vitamin B (Pyroxidine Hydrochloride),' J. A. M. A. 116:2484, May 31, 1941.

<sup>9</sup> Schlesinger, E. B. 'Curare: A Review of Its Therapeutic Effects and Their Physiological Basis,' Am. J. Med., 1:518, 1946.



commences within a few hours and may last for several days. Subsequent doses are given when the effect of the previous dose wears off. No toxic effects are observed when the drug is given in this manner.

### SENILE CHOREA

Senile chorea is a so called degenerative disease occurring after the age of 50, characterized by gradually increasing general choreiform movements. The patient usually has an unstable, irritable personality and often develops paranoid trends. At autopsy, senile plaques have been found in the putamen and caudate nucleus. It has been suggested that this is a form of Huntington's chorea that appears late in life.<sup>9</sup>

### MÉNIÈRE'S SYNDROME

Meniere's syndrome, characterized by attacks of true vertigo, with nausea and vomiting, and associated with tinnitus and progressive deafness, occurs at all ages, chiefly in the fourth, fifth and sixth decades of life. One third of the patients are over 50 at the time of onset of the disease. The condition warrants discussion because it is so frequently incorrectly diagnosed and in older persons is too readily attributed to a disturbance of the heart or arteries.

The syndrome is caused, apparently, by pressure or chemical changes in the endolymph with secondary involvement of the sensory end-organs in the semicircular canals and the cochlea.<sup>10</sup> The nature of these changes remains obscure, but

<sup>9</sup> Rothschild D. "Senile Chorea and Its Relation to Huntington's Chorea." *J. Mt. Sinai Hosp.* 5:517, 1938.

<sup>10</sup> Crowe S. J. "Meniere's Disease," *Medicine*, 17:1, February, 1938.

Atkinson<sup>11</sup> suggests that in a small proportion of cases allergic reactions may be responsible and that in the others, vasospastic disorders in the internal ear may underly the disturbance. Others have suggested that disorders of water metabolism or of electrolyte balance, particularly of sodium, may be the determining factors. All observers agree that the disorder bears no relationship to systemic disease to arteriosclerosis or hypertension or to otitis media. It is not due to cerebral arteriosclerosis.

The attack can occur at any time, even during sleep. In some cases increase in tinnitus or fullness of the head may warn of an impending seizure. In the attack the patient is seized with intense vertigo, all of the surrounding objects appear to revolve rapidly about him. Nausea and vomiting follow. The attack may last minutes, hours or days. In severe attacks the patient may be thrown to the floor by the violence of the vertigo and may even faint as a result of sudden drop in blood pressure. Under such circumstances the incorrect diagnosis of coronary occlusion is often made. At times the severe vasomotor disturbances may induce a coronary insufficiency of sufficient degree to cause cardiac infarction.

The patient is usually compelled to lie immobile, for movements of the body, particularly turning the head bring on renewed attacks. These attacks are not infrequently repeated and affect no

tests may show normal diminished or absent vestibular function. Audiometer tests reveal some impairment of hearing on the affected side. The side that is involved can usually be recognized by noting the ear in which tinnitus is experienced.

<sup>11</sup> Atkinson M. "Observations on the Etiology and Treatment of Menière's Syndrome." J. A. M. A., 118: 1753 Apr. 19 1941

Once the attack is over the patient feels quite well except that tinnitus usually persists. The spacing of attacks is variable; they may come very frequently or there may be remissions of months or years. Deafness is slowly progressive.

*Treatment*—Many forms of treatment have been suggested but all are difficult to evaluate because of the natural tendency to long remissions. Some have advocated restriction of fluid and sodium intake. Atkinson tests all patients with an intradermal test dose of 0.01 mg. of histamine dihydrochloride. If in response to this injection a large wheal with pseudopods forms, the case is considered allergic; attempts are made to eliminate possible allergens in the food or surroundings and desensitization with histamine is undertaken. The majority of patients do not fall in this category. These are treated with nicotinic acid, approximately 25 mg. intramuscularly daily at first, then at longer intervals. In the attack, the slow intravenous administration of 1.9 mg. of histamine acid phosphate in physiologic salt solution over a period of an hour and a half may induce a dramatic cessation of the seizure. I have found aminophylline or theobromine sodium acetate with phenobarbital useful. In patients who resist all treatment and in whom the attacks of vertigo are disabling, section of the vestibular nerve cures the vertigo but leaves the tinnitus unaffected. The operation may lessen the degree of deafness.

### GUILLAIN-BARRE SYNDROME

This is an infectious radiculoneuritis that occurs at all ages. In the young, recovery is the rule, but in older persons the disease is apt to be more severe and the mortality approaches 40 per cent.<sup>12</sup> The neurological disorder is usually

<sup>12</sup> Baker, A. B. "Guillain-Barre's Disease in the Older Age Group." *Geriatrics* 1:132, Mar-Apr, 1946.

preceded by an infection of the respiratory tract. Then polyneuritis develops with severe radicular pain, muscle tenderness and flaccid paralysis of the extremities. Facial nerve palsy is common. In older patients extension to the bulb with multiple cranial nerve palsies is frequent. The disease may take a myelitic form with bladder paralysis and rectal incontinence. There is little fever or leukocytosis. The findings in the spinal fluid are characteristic; there is an increase in the protein content without pleocytosis. Treatment is symptomatic.

### HERPES ZOSTER

Herpes zoster is more frequent in the young than in the old, but in older persons the postherpetic pain is apt to be exceptionally severe and persistent. Otherwise its clinical course does not differ in the various age groups. There is an apparent relationship between herpes zoster and varicella. It is not uncommon for a grandparent to develop zoster after one of the grandchildren has had chickenpox. I have also seen chickenpox develop in a child exposed to an older person who had herpes zoster. Herpes zoster is caused by a virus infection of the posterior ganglia. There is also a symptomatic form of zoster that develops when a posterior ganglion is invaded by a neoplasm or is the site of trauma or hemorrhage. Treatment of persistent root pain following the

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### MENTAL DISORDERS

discussed in Chapter 2. Physicians and laymen alike are too apt to attribute any and every mental disturbance in old people to organic regressive changes in the brain or to regard them as evidences of senile dementia. As a consequence, no treatment is attempted and mental deterioration progresses until an irreversible state is reached. It has been estimated that fully one quarter of patients with a diagnosis of senile or arteriosclerotic dementia can make social recoveries if given proper treatment. Psychoneuroses are common among the aged. The aging brain with its impaired blood supply has not the metabolic reserve power to withstand toxemia, nutritional lacks or severe circulatory disturbances and manifests its insufficiency by delirious reactions. Such disturbances may be superimposed on true psychoses or dementias. Thus it is vital to attempt an accurate diagnosis in every instance of mental disorder in the aged for, as we shall see, treatment often has gratifying results.

#### PSYCHONEUROSES

Bleuler has said: 'Senility often becomes a disease only as a result of the sudden cessation of the ordinary attractions of life. The changing social status of the aged forces them to make so many adjustments to their environment that it is not astonishing that many of them develop psychoneuroses. In some persons the neurosis repeats or continues the patterns of their younger years. The mental disturbance may be traced to the patient's inability to adapt to new situations, to the personal difficulties brought on by the declining years of life. Adjustment to these impacts depends in part on the habitual reactions built up in one's younger years.'

Sooner or later every elderly person must face the fact that his body is aging. There is a decrease in strength and in skills, the skin visibly becomes thin and wrinkled, the hair

gray and scanty, there may be impairment of sight and hearing of sexual power, and the woman becomes aware of the loss of her good looks. Some persons have a sudden realization of their failing bodily powers, some specific incident—the inability to perform some act formerly carried out with ease, failure to hold their own among their companions—drives home to them awareness of their waning powers. Handicapping illnesses may have a similar effect. Of equal, or possibly of even greater, importance is the impact of the cultural traditions of society. Loss of gainful work with stoppage of income, leads to dependence of the aging worker, to loss of his own home with a readjustment of his mode of life, often to enforced residence with his children as an unwanted guest. The aging person learns that he has become useless, that he is no longer an important member of society. Abrupt retirement from one's life work, which is becoming increasingly common in our industrial civilization involves a radical change in the habits and customs of living and in one's outlook on life. There is a loss of incentive and of routines of living that have been built up over the years. In many instances loss of home and of accustomed routine leaves the aging person with no privacy into which he can retreat.

As a result of all of these attacks by a hostile environment on the essence of his living the old person either fights for self preservation and develops aggressions or, finding the situation too difficult for his waning powers retreats into fantasy. He acquires anxieties. The mechanism of the neuroses in the aged does not differ from that in younger persons. Anxiety is commonly transferred to the visceral functions of the body. The aging person becomes absorbed in the natural operations of his digestive tract, develops a too great interest in his bowel habits or acquires dietary idiosyncracies because

of digestive symptoms. He may develop palpitation or other cardiovascular symptoms. These complaints, which may become hypochondriacal, are often used to maintain domination over his children and may be tools to give the children a sense of guilt—they are aggressive tendencies. Depression may develop, or there may be excessive fatigue. Other aging individuals may withdraw from life and evince little interest in their environment. They may develop exaggerated routines of living which may become compulsions. Lack of interest in what is going on about them in a world in which they no longer have a part contributes to their forgetfulness of recent events, and they find refuge in reminiscence. Small wonder that many are so lonely, tired and hopeless.

Many elderly men and women continue to have normal sex impulses, but because of the social environment and the general view that old people should be beyond such desires they find normal gratification difficult, they often resort to masturbation and this gives rise to a sense of guilt. The thwarted sex impulse commonly gives rise to anxiety or to hypochondriacal symptoms of low back pain and dysuria.

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cial problems, to preserving some sphere of economic usefulness and self sufficiency to its growing numbers of industrial derelicts. Only by social planning can the many real problems and difficulties of the aged be met, and only by some such means can many older persons be prevented from developing psychoneuroses. Treatment of the psychoneuroses must lean heavily on the social readjustment of the individual, on finding ways of granting him the needed self-expression, the security, the independence, the self respect which he requires. Both individuals and society should prepare years in advance for the age of retirement, to find some

means of making such retirement gradual of preserving some self sufficiency to the aging member When an old person is suddenly thrown in the discard it is too late to teach him how to meet the onslaughts of life by rebuilding his character He needs some routine some grooves of usefulness to guide his remaining years of life

# DELIRIOUS REACTIONS

A delirious reaction may be defined as a reversible psychosis marked by disorientation hallucinations and fear It may be the sole mental disorder or may be superimposed on a more serious mental disturbance or psychosis Included in the term are the so called exhaustion and toxic psychoses In old persons the cerebral circulation through the narrowed sclerotic arteries is often just adequate to maintain the normal function of the brain Any slight disturbance may upset the balance and lead to transient anoxemia or other damage to the brain cells Operation trauma infection heart failure cachexia sudden dehydration with mercurial diuretics and protracted administration of sedative drugs such as the bromides and barbiturates may induce delirious reactions At times prolonged malnutrition or diets lacking in vitamin content may induce a pellagra like psychosis The insecurity and the natural anxiety of the patient often form the basic psychological background of the delirious reaction

Delirious reactions are common in older patients Doty<sup>13</sup> encountered them in 15 per cent of patients over 40 seen as a psychiatric consultant in a large general hospital Robinson<sup>14</sup> reported that 20 per cent of all patients over age 60 ad

<sup>13</sup>Doty E J Incidence and Treatment of Delirious Reactions in Later Life *Geriatrics* 19:1 Jan Feb 1948

<sup>14</sup>Robinson G W Jr The Toxic Delirious Reactions of Old Age in Kaplan O J (ed) *Mental Disorders in Later Life* (Palo Alto Calif Stanford University Press 1945)



mitted to a neurological hospital had delirious reactions and that an additional 20 per cent had delirious reactions superimposed on some other type of mental reaction. Symptoms may come on suddenly, the patient may be drowsy in the daytime but soon grows restless, especially at night, and has frightening dreams. Soon he loses his orientation, becomes fearful and develops hallucinations and then delusions which may have a paranoid trend. Bodily tremors and incontinence as well as urinary incontinence develop, the patient becomes noisy and uncooperative. Unless vigorous treatment is instituted, dehydration and malnutrition develop and aggravate the mental disturbance. Death may follow. With a favorable outcome, the delirium may last from a few days to weeks.

*Treatment*—In the presence of complicating disease, the underlying condition should be treated. Infection should be combated, heart failure corrected and the diet made adequate. Feeding by stomach tube to assure a high calorie diet is often necessary. Intravenous administration of fluids to correct dehydration is important, and glucose and amino acids may be given by this route. Large doses of vitamins should be given, parenterally if necessary. Particularly important is nicotinic acid, which should be given in doses of 100 mg three times a day for a week or two. Secondary anemia should be treated with iron or, if necessary, with transfusion. Sedatives should be avoided as much as possible. The bromides and the barbiturates often aggravate the delirium. If quieting drugs are needed, morphine may be employed, or scopolamine in a dosage of 1/150 gr three times a day. Continuous baths may be quieting.

Intelligent vigorous therapy often cures the mental disturbance. It is well to remember that nutritional and toxic elements may superimpose delirious reactions on a tri-

and arteriosclerotic psychosis and that treatment such as that just outlined may salvage the mentalities of many of the aged who in former years would have been allowed to become senile derelicts. Robinson<sup>15</sup> reported 60 per cent complete recoveries among patients of 60 or more who were admitted to a hospital for mental diseases with severe abnormal mental reactions. Most of them had remained well when checked six months later.

### PSYCHOSES

Mental disorders become increasingly frequent with advancing years. A study in Massachusetts<sup>16</sup> showed that while about 2 per cent of the population between the ages of 30 and 60 in that state were admitted to mental hospitals, a much greater proportion of persons over 60 entered these institutions, the frequency increasing with each decade. Thus about 3 per cent of the male population of Massachusetts between the ages of 60 and 69, 5.5 per cent of those 70 to 79 and 8.5 per cent of those 80 to 89 years of age were admitted to mental hospitals. The frequency of mental disease in women is somewhat lower than in men, but the same increasing prevalence with advancing years is evident. Thirty per cent of the patients admitted to the state hospitals for mental diseases in New York State are over the age of 60. Not all of these suffer from senile or from arteriosclerotic dementia. There are those with toxic psychoses, with manic-depressive psychosis and with paresis. About 9 per cent of persons on first admission have senile psychoses, and again as many have psychoses secondary to arteriosclerosis of the cerebral arteries.

<sup>15</sup> Robinson, G. W., Jr. "The Abnormal Mental Reactions of Old Age." *J. Missouri M. A.* 39:36 February 1912.

<sup>16</sup> Dayton, N. A. *New Facts on Mental Disorders* (Springfield, Ill.: Charles C. Thomas, Publisher, 1940).

The chief psychoses encountered in older persons are the involutional psychoses, senile psychoses and psychoses with cerebral arteriosclerosis. Their relative frequency and age distribution are shown in Table 8.

TABLE 8—RATES OF FIRST ADMISSIONS TO ALL HOSPITALS IN NEW YORK STATE PER 100 000 OF GENERAL POPULATION OF SAME AGE 1942\*

AGES	SENILE PSYCHOSES		PSYCHOSES WITH CEREBRAL ARTERIOSCLEROSIS		INVOLUTIONAL PSYCHOSES	
	M	F	M	F	M	F
50-54	1.1	0.5	20.0	20.3	16.9	57.3
55-59	2.1	3.8	53.1	47.2	14.7	43.8
60-64	8.1	15.1	107.6	94.4	12.8	18.5
65-69	33.0	54.9	199.9	168.1	8.7	11.1
70 and over	254.9	311.1	333.8	264.1	0.8	1.4

\* Adapted from Pollock in Kaplan O. J. (ed.) *Mental Disorders in Later Life* (Palo Alto, Calif.: Stanford University Press, 1945).

### INVOLUTIONAL PSYCHOSES

Involutional psychosis or melancholia is three times as frequent in women as in men. Its age of occurrence is between the years 45 and 55 in women and about a decade later in men. Its cause cannot be found in the hormonal changes that accompany the climacteric, although in some cases these may contribute to the disorder. Apparently constitutional factors are of great importance. Persons who develop this psychosis have rigid, repressed personalities. They display obsessional characteristics, a surrender to meticulous detail. Environmental factors that become operative at the time of life when the psychosis develops may play a rôle in its development. The patient has reached a period when further advance is difficult; he may realize that he is a failure, he cannot adjust to financial reverses or other stresses such as the death of close relatives and friends.

Prodromal symptoms of depression, irritability and loss of weight, with feelings of unreality, and a tendency to withdrawal from the environment, may extend over several years. Finally the depression overwhelms the patient in full force, but it is associated with anxiety and with motor overactivity or agitation. Delusions of unworthiness and of sin and self-accusation with well marked suicidal trends are prominent. There are ideas of death, of poverty, religious delusions and auto erotic preoccupations. There may be somatic delusions, varying from hypochondriasis to the conviction that all of the organs have been lost. There may be little disturbance of consciousness and orientation. Paranoid trends may develop.

The psychosis runs a prolonged course and the outlook for recovery is not good particularly in persons over 55 years of age, in those with compulsive trends and in patients who in their youth exhibited great rigidity and narrow mental horizons. Approximately one quarter experience a spontaneous recovery, one fifth die by suicide or intercurrent disease and most of the others remain ill.

If the condition is recognized in its prodromal stages much may be done by helping the patient adjust to his environment, by correcting any existing physical disorders and possibly by substitution therapy with estrogenic or androgenic hormones. When the psychosis is fully developed, institutional treatment usually becomes necessary, particularly because of the risk of suicide. Full use should be made of hy

estrogenic hormone and with testosterone has been disappointing although it may be a useful adjuvant.

Today, electric shock treatment seems to hold out the best hope, and many good clinical results have been reported,

especially in patients with recent cases and in patients in whom schizoid personalities and paranoia are not prominent features. Age in itself is no contraindication to electric shock therapy unless there is severe cardiovascular impairment. Such treatment has a distinct field of usefulness in many patients past 65 who have depressive, manic or paranoid psychoses, conditions that ordinarily do not yield to other forms of therapy.<sup>16</sup> I have seen it bring about remarkable improvement in an 80 year old woman with a marked paranoid psychosis. Some few patients show intellectual deterioration as a result of treatment. Electric shock therapy should not be employed for senile dementia.

#### SENILE PSYCHOSES AND PSYCHOSES WITH CEREBRAL ARTERIOSCLEROSIS

Senile and arteriosclerotic psychoses and dementia are, by definition, caused by regressive or vascular irreversible changes in the brain. They are organically conditioned. Yet old persons who have died without any mental impairment may have brain lesions as severe as those seen in individuals who have died with senile or arteriosclerotic psychoses. There is no close correlation between the intensity of the mental symptoms and the extent of the cerebral pathological changes found at autopsy.<sup>17</sup> In this respect the brain resembles the heart and other organs, in which a close parallelism between structural and functional alterations can rarely be established. Just as the damaged heart compensates for its structural weakness and maintains the circulation, but becomes

<sup>16</sup> Feldman, F. et al. "Electric Shock Therapy of Elderly Patients," *Arch. Neurol. & Psychiat.* 56: 158, August 1916.

<sup>17</sup> Rothschild, D. "Senile Psychoses and Psychoses with Cerebral Arteriosclerosis," in Kaplan, O. J. (ed.) *Mental Disorders in Later Life* (Fals), Alto, Calif.: Stanford University Press, 1915.

insufficient if it is subjected to too great physical strain so the scarred and atrophic brain compensates and maintains a normal cerebral and emotional pattern for the aging person by passing its scarred structures and using other nervous pathways for co ordination Severe emotional shock, the death of husband or wife, the loss of home, any assault on the security and integrity of the individual may overtax these compensatory mechanisms so that a breakdown of cerebral function ensues Severe anatomical damage to the brain will induce mental disorders in anyone, but in most instances they are the personal and environmental factors overloading a damaged cerebral mechanism that lead to the mental breakdown

*Senile dementia*, characterized by a slowly progressive but inexorable intellectual impairment, becomes manifest, on the average at age 75 It is more frequent in women than in men The pathological picture is not pathognomonic, similar but less pronounced lesions are seen in the brains of aged persons who have died with their mental faculties intact There is a diffuse, but spotty cortical involvement with atrophy of the nerve cells Yet the cortical architecture is preserved There is a proliferation of the microglia and macroglia, and many senile plaques are evident

The psychosis usually develops slowly and manifests itself at first by a variety of neurasthenic complaints, weakness alterations in habits of sleep and growing irritability and emotional instability It may be precipitated by serious emotional traumas occasioned by the death of a close relative, loss of income or security or the development of some serious organic disease such as cancer Soon intellectual deficits become apparent the patient becomes forgetful, especially of recent events and his judgment and mental acuity are impaired Memory defects may lead the patient into dangerous

situations. He may wander from home and become lost, he may set himself or his house on fire by the careless handling of matches and stoves. There is diminution of attention and lack of sympathy for and understanding of others, the patient becomes self-centered, stubborn, depressed or euphoric. The old person exhibits in his psychosis his own lifetime personality, and many of his reactions are falsely attributed to old age. He lives in the past, may indulge in fabrications and be loquacious in his reminiscences. Defects in orientation develop, and eventually there is marked mental confusion, the patient becomes neglectful in matters of dress, eating and cleanliness. Loss of moral judgment may lead to asocial acts. Anxieties such as fear of poverty or of personal harm may develop. Some, especially persons who have had suspicious natures in their younger years, may exhibit a paranoid form of dementia. In these, there may at first be little impairment of memory or of intellectual functions, but eventually they, too, present the picture of progressive mental deterioration. Others exhibit periods of excitement or agitation or euphoria and may develop abnormal sexual activity. In time, deterioration becomes complete with progressive weakness, emaciation and incontinence of the sphincters. The onset of symptoms is 47 years and after admission to a mental hospital 17 year

*Arteriosclerotic dementia* bears no direct relationship to aging. It is the result of repeated cerebrovascular insults that lead to progressive destruction of brain tissue. Like arteriosclerosis, it is more common in men than in women. The average age at onset is 66 years, and the average duration of life after the development of symptoms is 34 years. The pathological lesion is characterized by vascular changes with

focal areas of destroyed brain tissue. Areas of softening and of hemorrhage are found especially in the basal ganglia and the temporo occipital regions. There are atherosclerosis of the larger cerebral arteries and arteriolosclerosis with hyaline degeneration of the smaller vessels. The patient commonly exhibits coronary and aortic sclerosis, hypertension, hemiplegia or other palsies, tremors and aphasia. In the psychoses with cerebral arteriosclerosis there is the same lack of correspondence between evident damage to the brain and mental symptoms that is encountered in senile dementia. Here too psychotic symptoms are more frequent and more prominent in persons who have previously had abnormal personalities.

The mental manifestations are much the same as those in persons with senile dementia, but the patient also exhibits symptoms and neurological signs resulting from cerebral injury. The psychosis is often precipitated by a cerebrovascular accident; there is sudden onset of confusion or delirium which may persist for weeks or months and which ends in death in half the cases. In others the mental disturbance subsides, but the patient complains of headache and dizziness, syncopal attacks or convulsions are common, and apoplectic seizures or attacks of heart failure recur. With each such episode there are further damage to the brain and further mental deterioration. Intellectual impairment, as a rule, is not as pronounced as in senile dementia, and there may be marked fluctuations in the degree of intellectual disturbance. Acute states of confusion or depressive symptoms are common in arteriosclerotic psychoses. Death results from the arterial disease, cerebral hemorrhage, renal insufficiency, or cardiac failure.

Treatment of senile and arteriosclerotic dementias is not very fruitful. It concerns itself with general hygiene, the



maintenance of nutrition and the establishment of a favorable environment. When there is marked depression or agitation or when the patient becomes a social problem because of his amoral actions or because of extreme untidiness, institutional care becomes necessary. Vigorous treatment of the organs damaged by the arteriosclerotic arteries, particularly treatment of cardiac and renal failure, has a favorable influence on the mental condition. If the blood pressure is high, phlebotomy is useful.

The *presenile dementias* present themselves chiefly in two forms.<sup>18</sup> Alzheimer's disease begins between 40 and 60, and although symptoms develop rapidly its course runs from two to 10 years. The clinical picture resembles that of senile dementia, but, in addition, there are aphasia, agnosia, apraxia, tremors and rigidity. Epileptiform seizures are common. The air encephalogram reveals diffuse cortical atrophy with enlargement of the lateral ventricles. The pathological picture resembles that of advanced senile dementia, but the so-called senile plaques are numerous and there are characteristic changes in the intracellular neurofibrils. Pick's disease may begin in the sixth or seventh decade and is often familial in its occurrence. It is characterized by slowly progressive dementia with focal cortical signs such as aphasia, agnosia and apraxia. The brain shows circumscribed areas of atrophy of the gray and white matter, confined to one or more lobes. During life, these atrophic areas can be recognized in the air encephalogram.

The suicide rate increases progressively with advancing years. One half of all suicides occur in persons 45 years of age or older, although this age group constitutes only one fifth

<sup>18</sup> Jervis G. A. 'The Presenile Dementias' in Kaplan O. J. (ed.) *Mental Disorders in Later Life* (Palo Alto, Calif. Stanford University Press 1945)

of the total population. The suicide rate per 100,000 in white males is 30 at age 40, 43 at age 50, 57 at age 60, 63 at age 70 and 80 at age 86. For females, the incidence is much lower and the progressive increase with the years is less evident. The rate per 100,000 at age 40 is 8, and between the ages 50 to 80 it remains fairly uniform at about 10.<sup>19</sup> Negroes have a much lower suicide rate than do whites.

Often medicolegal problems arise regarding the mental competency of the aged. This may relate to the making of financial transactions or, more often, of a will. Sexual transgressions such as exhibitionism or attacks on children, loss of a sense of values and lack of judgment leading to financial irregularities in persons of previous integrity may be first evidence of senile deterioration. It is important that the family practitioner make a written record of his observations of the mental reactions of his old patients, just as he does of their physical ailments. Such notes may be of great service if legal problems should arise.

<sup>19</sup> Dublin L. I. and Bunzel B. *To Be or Not to Be: A Study of Suicide* (New York City: Harrison Smith & Robert Haas, 1933).



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